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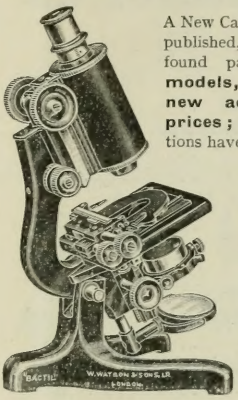

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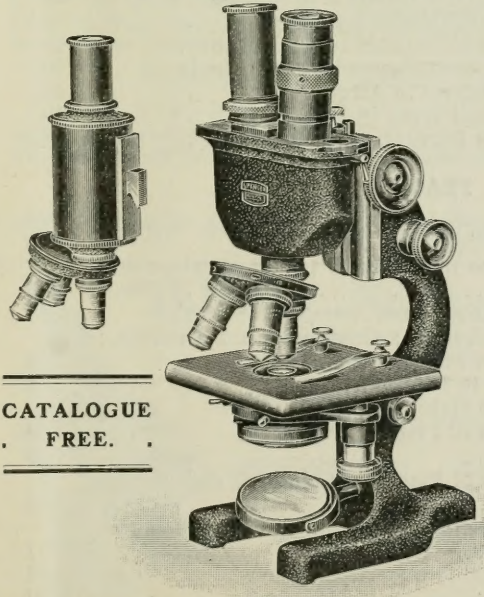

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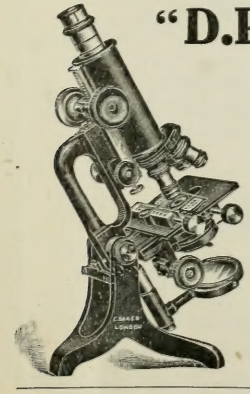

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BUREAU NOTICES.—*Continued.*

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PHILIPPINE JOURNAL OF SCIENCE. Series B. 1912, Vol. 7, No. 3; 1917, Vol. 12, No. 6.

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TROPICAL DISEASES BULLETIN.

Vol. 18.] 1921. [No. 5.

BERIBERI: A CRITICAL REVIEW.

By Surgeon Rear-Admiral Sir P. W. BASSETT-SMITH, R.N. (retd.), K.C.B., C.M.G., F.R.C.P., F.R.C.S.

Sectional Editor, Tropical Diseases Bureau.

No great advance has been made during the past few years in our knowledge of the etiology, pathology, or treatment of beriberi. The general consensus of opinion that the disease is only the evidence of an avitamin syndrome which includes a widely spreading group having rickets at one extremity and beriberi at the other, as was described by DARLING in 1915, has received constantly increasing proof.

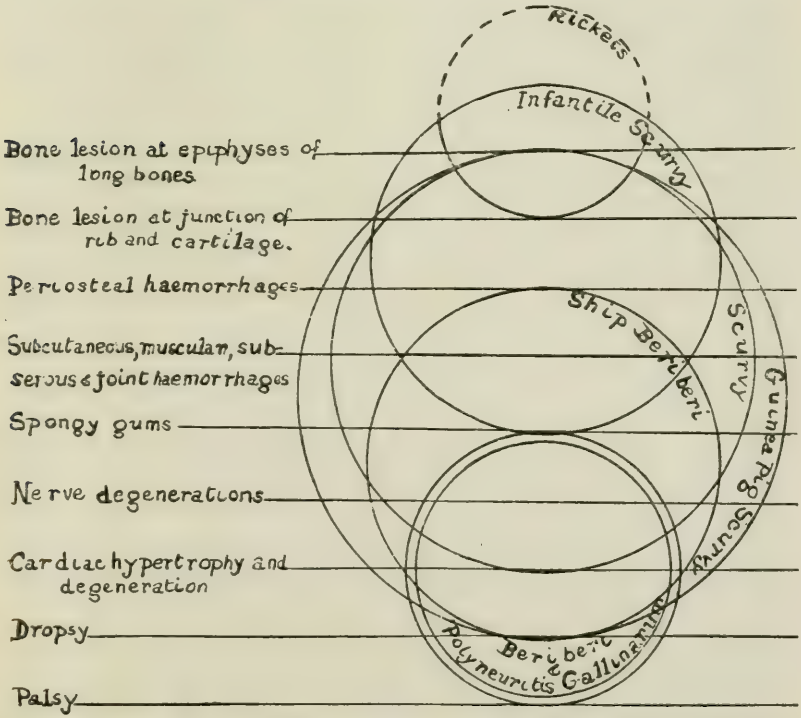

Chart showing affinities of the food deficiency syndromes [after DARLING].

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Endemic beriberi was originally considered to be a disease confined to the East in rice-eating natives, or to the crews of ships who had suffered great privation in protracted voyages. Through the researches of STANTON, FRASER and BRADDON, who proved the etiological importance of the various kinds of rice, and of HOLST with regard to the dieting of the seamen, the disease has now been placed under control as far as advice and medical instruction can place it, but year after year evidence is obtained of the wide geographical distribution of its endemic forms.

Cases are reported from the Congo region of Africa among manioc eaters by VAN DEN BRANDEN, from the east of Belgian Congo by WYDOOGHE (J.), and from Madagascar by CLAIR (F.); in Brazil by FRAGA (C.), LINDSAY (J.), and RODRIGUES (F.). The latter author stated in 1920 that beriberi was still of great importance to the Brazilian Navy and was common in the northern part of Brazil; he had no doubt the disease in this country was the same as that recognized in the East. From Cuba and Porto Rico cases are reported; in the latter island, RIDDELL (J.) found that in one regiment there were 56 cases and two deaths and that the disease was distinctly due to food factors. FERNANDEZ MARTINEZ found a poor peasant of Alpujaras with a definite beriberi syndrome produced by a deficiency diet, which was cured by correcting the food; though this is the first case reported from Spain, it is probable that in Castile and in the Basque provinces others would be found if carefully looked for. These instances, with what was previously known, show that beriberi is not dependent upon geographical position, nor on the use of rice as food.

During the War, 1914–18, and since, two groups of cases have been mentioned: (1) in the Mesopotamia force: (2) in southern France among the Indo-Chinese labourers. In the first, at the siege of Kut, the disease was limited to the Europeans, whose ration was biscuit and white bread, with tinned meat; later, horseflesh and brown bread (half white flour and half atta) were issued, and the incidence fell: 155 cases were admitted to hospital, and there was a mortality of 15 per cent. Practically all the cases were of the "wet" type. Starvation and malaria were undoubtedly predisposing causes. SPRAWSON (C. A.) divided the beriberi cases in the Mesopotamian forces into three groups: (1) British cases among the landed troops, (2) Chinese cases among those landed, (3) ship cases from the Indian crews; he was in favour of an infective theory for those on shore, but the condition of the food as taken rather than as rationed was not sufficiently considered. The second group of cases, among the Indo-Chinese, Chinese and Africans landed in France, was described by SICARD (J. A.), LEGGATE (A. R.), ROGER (H.) and MARCHOUX (E.) It is generally stated that these men, after landing, were on a well-considered full diet, equal to that of the Europeans; yet the disease developed among them. Many cases were undoubtedly contracted during the passage and were acute in character; others were latent forms developed during changed conditions of work, and after an *influenza outbreak*, which lowered resistance. SICARD and ROGER, from a large experience at Marseilles (228 cases), state that in no instance could a spread of infection from the sick to the healthy be traced, and that there was no danger of conveying the disease to the indigenous population by landing the cases; they note that the supposed epidemics in France described by CHANTEMESSE and RAYMOND were more like scurvy than beriberi.

From a *pathological* point of view little has been done except by the Japanese. SICARD in three post-mortems made at Marseilles found

congestion and haemorrhage in the stomach and intestines, with degeneration of the Schwann areas of the large nerves, but without interstitial neuritis.

KIMURA (O.), in a monograph on the nerve changes in beriberi, shows that these are degenerative and not inflammatory. The first perceptible signs of degeneration appear in the axis cylinders, but peri-axial changes also occur; the destructive products are absorbed locally, and regenerative processes are found by the side of acute degenerative changes. Primary damage of the central nervous system may occur in acute cases. The hypertrophy and dilatation of the heart are not constant and essential characters, but in prolonged experimental cases the muscle fibres show marked fatty changes.

HONDA (K.), from 45 post-mortem examinations, describes changes in the nervous system, intestines, muscles and bones. The vagus and sympathetic nerves are much less affected than the spinal nerves, and in the peripheral nerves the coarse fibres degenerate much more than the fine fibres. The changes in the ganglionic cells of the spinal cord were much less marked than they are supposed to be, and do not advance beyond a degree that can be quickly recovered from, the changes being partly toxic and partly secondary ascending. It is the active degeneration of the Schwann cells in the sheath that causes the destruction of the nerve fibres; these degenerative masses act as foreign bodies and an irritant to the adventitious cells, which increase in number and wander into the perivascular spaces. Fatty degeneration of the muscle fibres does not run parallel with the nerve changes and is toxic in origin; the susceptibility of the heart's action is mainly due to the structural changes in the cardiac muscles, a direct result of the toxin.

In infantile beriberi he states that the loss of voice is undoubtedly due to definite changes in the nerves and the muscles supplied by them.

Thus with both authors the pathological evidences are in favour of a direct toxin and secondary nerve changes. With regard to the question whether there is an increased pressure of the cerebro-spinal fluid, the results are conflicting. SICARD found none, but OHIDA (H.) registered in acute cases a pressure as high as 280 mm. and as low as 100 mm. in slight ones. MUIRA has noted that when the cerebro-spinal tension is high the blood pressure may be low. This high pressure may account for the vomiting in late severe cases and is always of bad prognosis.

Both in man and in experimental beriberi an increase in size of the suprarenals, through congestion and true hypertrophy, has been found by SHIMBO (M.), MIDORIKAWA (K.), OHNO (S.) and MCCARRISON (R.) with an increased adrenalin output, as is also found in scurvy; the other glands of internal secretion are generally atrophied. Various evidences of changes in metabolic activity have been noted.

ARIMA (E.) found an increase in the non-protein nitrogen content in the blood, from 45 to 65 mgm. as compared with that of healthy Koreans, which was 28 to 37 mgm.; in uraemia 200 mgm. may be registered. The increase appears to be due to a disturbance in the balance between the kidney excretory function and protein katabolism, and not to cardio-vascular failure.

SUZUKI (T.) has also shown that the nitrogen metabolism of infants is distinctly injured. YOSHIKAWA (I.), YANO (K.) and NEMOTO (I.) found an increase of urea in the blood from 0·06–0·08 in health to 0·07–1·36 in beriberi. Of 11 cases tested, the two with the highest

coefficients died. It is possible that the cardiac failure in beriberi is due to the accumulation of some unknown product in the blood, the elimination of which is coincident with the elimination of urea.

Suzuki (T.) found a hypoglycemia in nursing beriberi infants, and considers this to be due to an insufficient adrenal function. Suga (T.), on the contrary, found in acute cases an increase of blood sugar, which decreased during convalescence, and Funk (C.) described such an increase in experimental avian beriberi. Findlay (G. M.), from experiments on birds, shows that there is evidence of nuclein degeneration with chromatolysis of the cells of the central nervous system with a retention of lipoids in the adrenal cortex, that the nucleic acid of the brain and liver of affected birds was decreased, and that the administration of vitamin B to birds suffering from beriberi causes a removal of the lipoid from the adrenal cortex, an increase in the nucleic acid content of the brain, with the reappearance of Nissl's granules, and disappearance of the paralytic symptoms. From this experimental evidence he deduces that vitamin B is an essential factor in the synthesis in the body of animal nucleic acid.

Kawakami (D.) has produced beriberi in goats by feeding them on polished rice ; the pathological changes were similar to those found in other animals.

Etiology.—There have been a few reports in favour of a toxic germ being introduced in diseased rice, but none is convincing, and there is much evidence against the infective nature of the disease. That it is dependent upon a deficiency of the vitamin B in the food is certain, whether this be rice, white flour, manioc or other carbohydrate, but how this acts is still debatable. The theory of Vedder (E. B.) that the vitamin is a necessary " building up " factor for the nervous system is strongly opposed by Funk, Braddon, Cooper and Walshe and others, and Vedder in turn disagrees with those who hold the belief that an excess of carbohydrate intake influences the amount of vitamin required.

The purely negative deficiency theory is now to a great extent held to be insufficient, and this is ably discussed by Walshe (F. M. R.), who believes that it is the defective metabolism of carbohydrates brought about by the deficiency of the vitamins which produces a toxic by-product or *positive* factor as yet unknown (probably an enzyme), and causes an intoxication chiefly affecting the nervous system. This conception is compatible with the food deficiency view and reconciles the toxic theory with it. The three chief characters of beriberi are nervous, cardiac, and oedema. Walshe holds with the pathologists that the nervous lesions in beriberi are not specific to the disease, for they cannot be distinguished from those of toxic polyneuritis ; the cardiac changes are similar to those found in diphtheria, and the oedema is attributable to changes in the suprarenals and their output. Lumière (A.) considers that in pigeons the vitamin acts as an excito-secretor, and maintains the tone of the organs of digestion. Weill (E.) and Mouriquand (G.) experimentally showed that the paralysis in the acute form is functional, and that definite pathological lesions, such as are found in human beriberi, can only be produced in chronic forms induced in pigeons by special feeding.

The whole subject of the deficiency diseases is one of vast importance and of wide range. The special accessory substances known as vitamins are three :—(1) A fat-soluble vitamin A, which is necessary for growth ; (2) A water-soluble vitamin B, or anti-neuritic factor, which is fairly thermostabile—namely, can withstand 100° C. for two

hours; and (3) a thermolabile vitamin, or anti-scurvy factor. These facts are well demonstrated, and the distribution of the vitamins in foods has been placed on a practical basis through the work of CHICK and HUME at the Lister Institute, and others. The deficiency diseases are so closely associated that a thorough knowledge of the whole group is essential in the study of the subject. MCCARRISON, in India, has carried out some most valuable work on birds and monkeys. He considers that want of vitamins, when associated with a diet too rich in starch, leads to a disordered function of the whole endocrine system, and that this disordered endocrine function leads in turn to imperfect carbohydrate assimilation, disturbance of carbohydrate metabolism, and resultant muscular atrophy. The whole digestive system is very easily disordered by an unnatural diet, and, by this, bacterial invasion of the gastro-intestinal tract and bacterial migration are facilitated. It is probable that there is an *infective* factor at work in many cases described as beriberi, in addition to the essential dietetic factor. He also concludes that a deficiency of certain accessory food factors gives rise to a greatly increased production of adrenalin, and this excessive amount of adrenalin is concerned in the causation of oedema found in many cases.

Ship beriberi holds a place intermediate between true beriberi and scurvy, and it is closely related to the disease found in the Rand miners of South Africa. In cases in the British Navy the development is often very slow, the consumption of rice is not an etiological factor, and the loss of the knee reflex with evidences of peripheral neuritis may not be well marked. HOLST found that its increased frequency in Norwegian ships coincided with three changes of food:—(1) Replacement of salt meat by tinned foods; (2) reduction of the pea ration; (3) replacement of biscuit by soft bread, usually made from white flour. Each of these changes caused a reduction in the anti-neuritic factor. He states that the disease was often *cured by fresh vegetables, potatoes and fruit,* but not always · beer, eggs and oats were also useful.

With regard to the cure of beriberi, the substances which have been found to have prophylactic value are here indicated. In Mesopotamia the use of yeast extracts or "marmite" was very beneficial, and in infantile beriberi "tiqui-tiqui" is specific. SALEEBY (N. M.) found that the hydrolysed yeast extract was more efficient in the infantile disease than the autolysed extract. SHORTEN (J. A.), in experiments with fowls, in which beriberi had been produced by feeding them on Rangoon rice, found that a daily ration of 5 gms., dry, of sun-dried carrots, onions, spinach and cabbage afforded complete protection, and that the vitamin content of these substances was relatively high. He states that these preparations will form a safe substitute for fresh vegetables in a ration for issue to troops.

This statement, at least as regards the prevention of beriberi, must, I think, be received with considerable reserve and requires further proof, as CHICK and HUME give the anti-beriberi value of *fresh* cabbage as nil, and that of onions and carrots as very slight. All authors agree that wheat-embryo, pulses, and yeast, have the highest anti-beriberi values, and that in these the vitamins are not destroyed at a temperature of 100° C., but at 120° the reduction of their activity is very rapid. HARDEN (A.) and ZILVA (S.) have attempted to isolate anti-neuritic substances, mainly in pyridine derivatives, but without any great success. FUNK (C.) and DUTCHER (R. A.) have experimented with

thyroid gland and other substances known to influence carbohydrate metabolism ; their experiments are instructive, but at present of little practical utility.

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DYSENTERY: A REVIEW OF THE LITERATURE OF THE LAST SIX YEARS.

By P. H. Manson-Bahr, D.S.O., M.D., M.R.C.P., D.T.M. & H., Cantab.
Sectional Editor, Tropical Diseases Bureau.

Part II.—Amoebiasis.

1. *Epidemiology.*

Amoebic Dysentery and Hepatic Abscess.—Prior to the outbreak of war in 1914 amoebiasis was generally regarded as a disease peculiar to the tropics and subtropics, occurring solely as an endemic infection in countries with a mean temperature of 60° F. or over.

Possibly, as Dobell suggests, the non-recognition of protozoal diseases in Great Britain may be ascribed to a habit of regarding intestinal protozoa as being peculiar to the tropics, so that search for evidence of protozoal disease in temperate countries is unnecessary.

With the advent of war the situation completely altered; from a comparatively few protozoologists trained in intestinal protozoology a new school sprang up, which became more powerful and numerous as the contest dragged on. The stimulus thus given to the subject resulted in the compilation of a large number of statistics, which have been collected and published by Dobell, his assistants, and a number of workers from the Liverpool School of Tropical Medicine (especially Matthews and Malins Smith).

Although a few isolated cases of intestinal amoebiasis and hepatic abscess had been noted before 1914, it was not till 1916 that an acute case of indigenous amoebic dysentery, complicated by hepatic abscess, was studied by Wenyon in the London Hospital (quoted by Dobell); subsequently another acute dysenteric case was recognized by Worster-Drought and Rosewarne. Later it was noted that "healthy" excretors* of the cysts of *Entamoeba histolytica* were comparatively common, and that about 3 per cent. of the indigenous civil and military population harboured the protozoa; in children 1·8 per cent. and in asylum patients 9·7 per cent. were found infected. In France an acute indigenous case was reported first by Orticoni and Ameuille; Dargein recorded an undoubted hepatic abscess, whilst Ravaut and Krolunitski calculated the proportion of healthy carriers of *Entamoeba histolytica* amongst the French population as high as 5 per cent. Amongst British troops returned from France in 1916–17, no less than 8 per cent. were found to be carriers. Since that date it has come to be recognized that the disease "amoebic dysentery" was comparatively common on the western front, though the proportion of these cases to the total number of "clinical dysenteries" was relatively small, probably less than 1 per cent.

In Holland also, *E. histolytica* occurs as an indigenous infection, and Kuenen records a number of amoebic dysentery cases as well as excretors of the cysts; Fischer apparently has found the same in Germany. Amoebic dysentery has recently been found indigenous in Spain, Italy and in Australia.

* Dobell has pointed out the inaccuracy of the term "cyst carrier." [Dobell & O'Connor. The Intestinal Protozoa of Man. p. 7.]

In the Eastern theatres of war amoebic dysentery was a common disease; it was probably more widespread in Mesopotamia than elsewhere; thus LEDINGHAM noted an average high rate of infection, ranging in 1918 from 14·24 per cent. of British troops up to 27·9 per cent. of Indian troops employed in that area. In Salonika, Egypt and Palestine, amoebiasis was always present, though at no time did it rage in epidemic form. This disease constituted from 5 to 10 per cent. of all the clinical dysenteries evacuated to hospitals. The "carrier" rate in British soldiers and in the native population was found to vary considerably in different war zones and will be considered subsequently.

An important point bearing upon the epidemiology of amoebiasis has been the discovery of the *Entamoeba histolytica* as a natural infection amongst animals. EICHHORN and GALLAGHER (1916) in America have recorded an outbreak of spontaneous dysentery amongst captive spider monkeys (*Ateles ater*), two of which developed hepatic abscess; DOBELL considers that the cysts of entamoeba in the faeces of *Macacus rhesus* are indistinguishable from those of *E. histolytica*, so that it is by no means impossible that monkeys may constitute a reservoir of infection.

The dog, like the cat, is susceptible to laboratory infection with *E. histolytica*; but amoebiasis probably does occur amongst them under natural conditions. WARE has recorded an outbreak amongst fox hounds in India, FISCHER a case of intestinal ulceration in a dog in China in which entamoebae were demonstrated; BAUCHE and MOTAIS a similar case in a dog recently imported from Cochin.*

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† References are to summaries in the *Tropical Diseases Bulletin* unless otherwise stated.

2. *Etiology.*

Dobell has published the most accurate description of *Entamoeba histolytica* and its life-history which has yet appeared, in his great work, "The Amoebae Living in Man" (1919). Its active forms vary greatly in size, ranging from 18μ to about 40μ. When at rest it assumes a spherical form, the protoplasm consisting of two well-marked layers—an outer clear, structureless ectoplasm, and an inner finely and uniformly granular endoplasm. Ingested red blood corpuscles in various stages of disintegration are often seen, but *bacteria and other inclusions* are not present in healthy specimens; vacuoles, resembling bubbles, indicate that the individual entamoeba is degenerate. The nucleus is not distinct in a perfectly healthy entamoeba, but in the earlier stages of degeneration it becomes visible; it is situated in the endoplasm, measures 4–7μ in diameter, possesses a small central karyosome, and is eccentrically placed.

The nuclear characters of *E. histolytica* (Fig. 1)—according to Dobell, a uniform layer of small chromatin granules at the periphery, the central karyosome with its halo and the absence of chromatin in the intervening space—serve to distinguish *E. histolytica* from all other intestinal species of the genus *Entamoeba*.

The life-history of *E. histolytica* in man appears to be as follows:—The active entamoebae live in the gut wall, where they multiply by division; subsequently a certain proportion migrate from the ulcerated surface into the lumen of the bowel and encyst, passing out ultimately with the faeces. Encystation occurs only in the bowel, for Dobell has shown that precystic amoebae after being passed out in the stools are unable to complete their development.

The precystic amoeba, which is smaller than the parent forms, comes to rest, becomes rounded and secretes a cyst wall. The *cysts* are smaller than the precystic forms. Primarily uninucleate, they subsequently become, when fully mature, quadrinucleate. They vary greatly in size; Dobell and Jepps have described five races of *E. histolytica* distinguishable by the size of the cysts, the respective mean diameters being 6·6μ, 8·3μ, 11·6μ, 13·3μ and 15μ. It is probable that the smaller races of cysts have been long overlooked.

The encysted organism forms in its cytoplasm blocks of a highly refractile substance giving all the reactions of chromatin—termed *chromatoid bodies* by Dobell; at the same time a vacuole of varying size forms in the cytoplasm. An iodophilic substance, probably glycogen, is generally present in the vacuole. When the cyst has reached the quadrinucleate stage no further nuclear divisions occur; the glycogen vacuole disappears and the mature cyst stains uniformly pale brown with iodine solution.

The cysts of *E. histolytica* survive for several weeks outside the body, if kept moist and cool. Some remain alive much longer than others, and as they die they become permeable to aqueous stains such as eosin.

Dobell considers that *E. histolytica* may sometimes form supernucleate cysts of eight nuclei, but that this happens very rarely.

When the cysts are swallowed by man they probably hatch in the small intestine, liberating small amoebae, which pass into the large bowel. The exact manner of excystation is still a matter of dispute. Chatton has found that the cysts, when swallowed by a cat, pass through its stomach without undergoing any marked change and, according to his observations, normally hatch in the small intestine, liberating young quadrinucleate amoebae which first live on bacteria,

but subsequently establish themselves in the tissues of the large gut. Penfold, Woodcock and Drew have claimed that they were able to hatch cysts by placing them in liquor pancreaticus. They observed but a single small amoeba emerge through a small aperture in each cyst. These experiments were subsequently repeated without success by Dobell and A. C. Stevenson, so that the matter can by no means as yet be considered settled.

Normally no other stages than those described above occur in the life-history, but exceptionally the entamoebae may migrate from their primary site of infection via the portal vein into the liver or brain, where they may give rise to abscesses. The organisms in these secondary infections are always typical tissue-inhabiting forms, precystic individuals and cysts being found in the intestinal contents only.

Cultivation of *E. histolytica* on artificial media has not yet been unquestionably successful. Cutler claims to have obtained cultures of pathogenic amoebae on six occasions out of forty-five attempts in an egg-broth and a blood-clot medium; sub-cultures had to be made daily. This work has been repeated with unsuccessful results by Dobell and Douglas and as yet has not been confirmed by other workers.

Four other species of intestinal amoebae are now recognized as parasitic in the alimentary tract of man, and it is necessary to mention them, as their differentiation has been accurately determined only during the period under review.

Entamoeba coli (Fig. 2) is a harmless commensal living in and feeding upon the contents of the large intestine, but never invading the tissues of its host. It is a large species, though subject to great variation in size. Its mean diameter is from 20 to 23μ, the extremes from 18 to 40μ. There is no marked differentiation of the protoplasm as in *E. histolytica*, and the granular cytoplasm generally contains food vacuoles and ingested bacteria, but never red blood-corpuscles or other tissue elements. The cysts exhibit considerable variation in size (between 10 and 30μ), and it is generally agreed that *E. coli* is a composite species consisting of a number of races. The immature cyst is uninucleate, the fully mature cyst octonucleate. Chromatoid bodies, though present in young cysts, usually disappear when they are fully mature.

Endolimax nana (Fig. 3) is a small non-pathogenic amoeba 6 to 12μ in diameter, possessing a characteristic vesicular nucleus and a large irregular eccentric karyosome. It probably inhabits the small intestine and lives upon its contents. The cysts are oval bodies having characteristic features, and are about the same size as are the active forms. When mature they contain four nuclei and a few refractile granules, no chromatoid bodies, and a considerable amount of glycogen.

Iodamoeba bütschlii (Fig. 4) is a small amoeba intermediate in size between the two former species and measuring 9 to 20μ in diameter. The cytoplasm contains food vacuoles, bacteria and other food particles. The nucleus is vesicular, with a moderate-sized central karyosome. This amoeba feeds mostly upon the micro-organisms of the large intestine. The cysts are uninucleated structures, frequently irregular in shape, and measure 9 to 12μ in diameter. They are conspicuous mainly by the large dense glycogen mass they contain, which stains intensely with iodine. The nucleus of the cyst is eccentrically placed and contains a conspicuous compact karyosome.

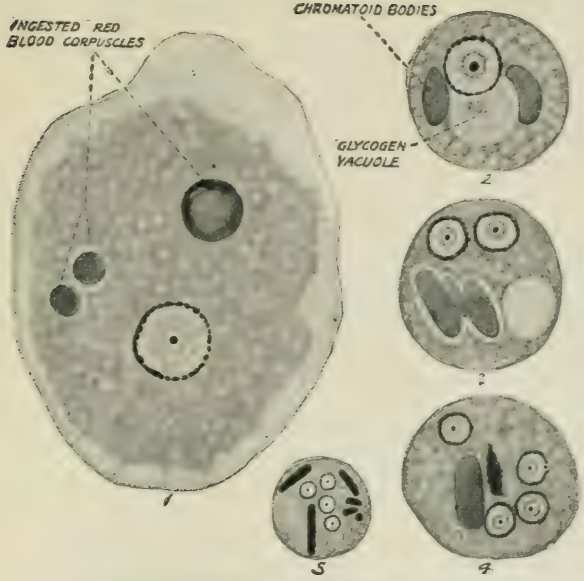

Fig. 1. *Entamoeba histolytica.* ×2,500 diameters. (After DOBELL.)
1, Active amoeboid form with ingested red blood-corpuscles; 2, Uninucleate cyst; 3, Binucleate cyst; 4, Quadrinucleate cyst; 5, Quadrinucleate cyst, small race, 6·6μ in diameter.

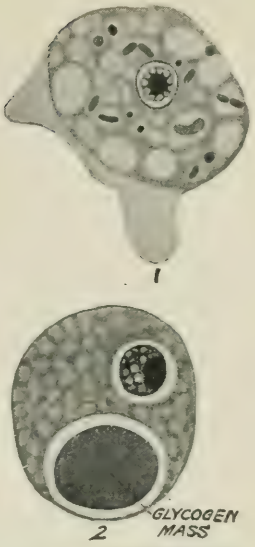

Fig. 4. *Iodamoeba bütschlii* ×2,500 diam. (After DOBELL.)
1, Active amoeboid form with ingested micro-organisms; 2, Mature cyst, "iodine cyst," containing large glycogen mass.

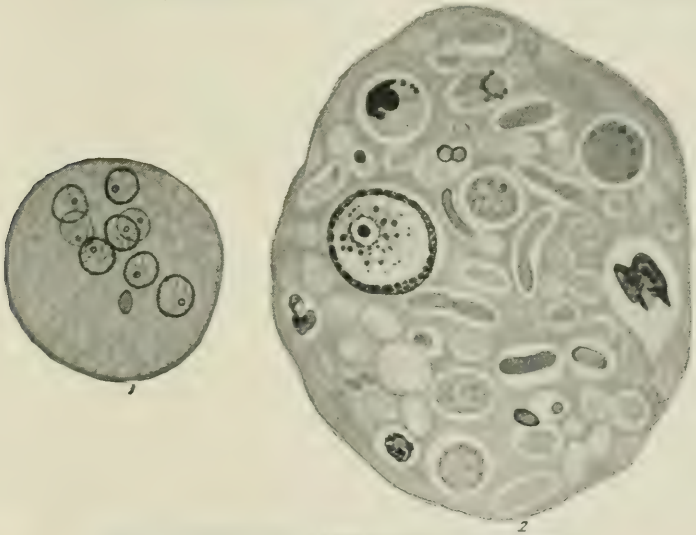

Fig. 2. *Entamoeba coli.* ×2,500 diameters. (After DOBELL.)
1, Cyst with eight nuclei; 2, Active amoeboid stage, with ingested food material.

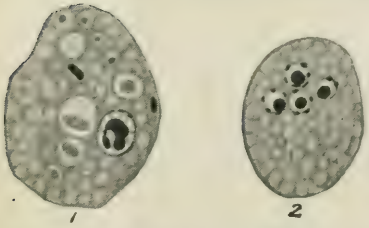

Fig. 3. *Endolimax nana.* ×2,500 diam. (After DOBELL.) 1, Active amoeboid form; 2, Quadrinucleate mature cyst.

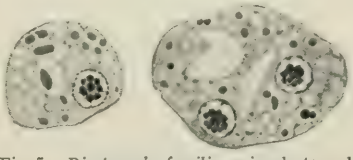

Fig. 5. *Dientamoeba fragilis,* uninucleate and binucleate forms. ×2,500 diam. (After DOBELL.)

The Intestinal Amoebae of Man.

[Reproduced by permission from "MANSON'S TROPICAL DISEASES."]

Dientamoeba fragilis (Fig. 5) is a very small and rare amoeba of the large intestine. Its usual diameter is about 9μ. Binucleate individuals are frequently encountered. The nucleus is large, spherical and vesicular, the karyosome being large and composed of numerous granules. The organism lives exclusively upon bacteria; no cysts have as yet been found.*

3. *Pathology.*

Entamoeba histolytica penetrates the tissues by destroying the cells; probably, as DOBELL suggests, it does not force its way into the tissues, but secretes a powerful cytolytic ferment which dissolves them, so that these organisms come to lie in pools of broken down tissue which they absorb as nutriment. In the base of the ulcer they also ingest red blood corpuscles and cell-fragments, but by no means to the same degree as they do when free in the bowel contents. LYNCH believes that the entamoebae gain entrance to the deeper tissues through the glands of Lieberkühn, and in the earlier stages of penetration form a nest at the base of a crypt; thence they invade the submucosa. The port of entry once having been established, they migrate for a considerable radius in all directions, causing thrombosis of the blood vessels of the submucosa and muscularis with consequent necrosis of the superjacent tissues and the formation of a typical ulcer. According to JOB and HIRTZMANN secondary bacterial infection of the ulcers invariably takes place.

BARTLETT has given a very good description of the morbid anatomy and histology of amoebic ulceration of the large intestine, with typical "flask-shaped" ulcers as he terms them.

MANSON-BAHR considers that amoebic ulcers may be differentiated by their gross characteristics from those caused by a chronic bacillary infection by the following features:—Amoebic ulcers commence usually in the solitary lymphoid follicles and run parallel to the long axis of the gut; they are discrete, oval in outline, flask-shaped in section, involving mucous membrane, submucosa and muscular coats of the intestine. They are distributed throughout the large intestine from the caecum to rectum, being usually larger and more extensive in the lower part; the small intestine is never affected. The mucous membrane between the ulcers appears unaffected.

Chronic bacillary ulcers, on the other hand, appear to commence on the free folds of mucous membrane and to be distributed transversely to the long axis of the gut; in outline they are serpiginous, with ragged undermined edges often intercommunicating with neighbouring ulcers, and involving mucous membrane as well as muscularis mucosae. They are found in the last two feet of the ileum as well as in the large gut. The intervening mucous membrane is inflamed and presents an unhealthy plush-like appearance.†

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* REFERENCES.

SELLARDS (A. W.) & BAETJER (W. A.), vol. 5, p. 204; PENFOLD (W. J.), WOODCOCK (H. M.) & DREW (A. H.), vol. 8, p. 116; WENYON (C. M.) & O'CONNOR (F. W.), vol. 11, p. 43; CHATTON (E.), vol. 11, p. 251; DOBELL (C.) & JEPPS (M. W.), vol. 12, p. 4; CUTLER (D. W.), vol. 12, p. 292; DOBELL (C.), "The Amoebae Living in Man," 1919, John Bale, Sons & Danielsson, Ltd.

† REFERENCES.

JOB (E.) & HIRTZMANN (L.), vol. 9, p. 176; BARTLETT (G. B.), vol. 10, p. 146; MANSON-BAHR (P. H.), vol. 14, p. 320; LYNCH (K. M.), vol. 17, p. 6.

4. *Symptomatology.*

Cannata has drawn attention to the occurrence of amoebic dysentery in children of tender years in places where the disease is endemic. Cases are recorded in children between 2 and 15 years of age.

Dobell and Stevenson have given accounts of cases in which infection with *E. histolytica* appears to have been prolonged; the infection may persist certainly for 16, it may be for 34 years. The capacity for latency which this infection may exhibit has certainly struck most observers. Relapses have occurred in individuals who have for five or even six years remained free from all evidence of the disease. We know from the investigations of Wenyon and O'Connor and the Liverpool workers that the majority of the excretors of *E. histolytica* cysts give no history of intestinal disturbance, and that, in fact, the percentage of post-dysenteric cyst-passers is about equal to that of those with no previous dysenteric record. It may be, as Dobell suggests, that not more than 10 per cent. of persons who become infected with *E. histolytica* ever suffer to any appreciable extent from their infections.

It has come to be recognized that a large proportion of recurrent diarrhoeas are amoebic in origin, and to this condition, in which true dysenteric symptoms may never appear, the name "amoebic diarrhoea" should be applied.

Hepatic and Brain Abscess.—Stout and Fenwick record the association of liver abscess with a similar accumulation of pus in a cavity "the size of a pigeon's egg" in the brain; active *E. histolytica* were present.

Armitage has described a similar case in which the organisms were numerous. He has collected evidence of 48 cases altogether from the literature: only three have been in women.

Several papers have appeared on the subject of liver abscess adding little to existing knowledge.

Talbot has summarized the signs and symptoms as follows:—

(1) General enlargement of the liver.
(2) Pyrexia, either continuous or remittent.
(3) Leucocytosis of 18,000.
(4) A local tender spot over the liver.
(5) Signs of irritation at base of right lung.
(6) Rapid wasting.
(7) Heavy nocturnal sweats.

Symptoms of less value are:—Local bulging, pallor, rigors, referred pain in right shoulder, rigidity of right rectus.*

5. *Diagnosis.*

In the acute stage of the disease Ledingham has pointed out that fully 90 per cent. of untreated amoebic dysenteries may be correctly diagnosed by a single microscopical stool examination. The amoebae may be recognized as highly refractile organisms often

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* References.

Symptomatology.—Cannata (S.), vol. 8, p. 437; Dobell (C.), vol. 9, p. 178; Smith (A. M.) & Matthews (J. R.), vol. 11, p. 47; Dobell (C.) & Stevenson (A. C.), vol. 12, p. 3; Brug (S. L.), vol. 18, p. 34.

Hepatic and Brain Abscess.—Stout (T. D. M.) & Fenwick (D. E.), vol. 12, p. 290; Armitage (F. L.), vol. 14, p. 93; Gibson (C.), vol. 14, p. 316; Talbot (P.), vol. 15, p. 191; Candler (A. L.), vol. 16, p. 2; Ludlow (A. L.), vol. 17, p. 8.

showing active movements and emitting with explosive suddenness pseudopodia as lobes of clear ectoplasm. Under favourable conditions they show a rapid, progressive flowing movement. The most important characters for identification of *E. histolytica* in unstained preparations are these active explosive movements and the frequent presence of ingested red blood corpuscles and absence of other inclusions; in fact, as WENYON has pointed out, the presence of ingested erythrocytes in a motile amoeba is diagnostic. As the active entamoebae rapidly die and disintegrate after leaving the intestine, it is essential that the examination should be made soon after passage of the stool, within two hours if possible. It is also important that the bed-pan or other utensil should be free from antiseptics and dry, as even water or urine rapidly kills these organisms.

The *macroscopic appearance* of an amoebic dysentery stool during the acute stage affords a probable diagnosis. In brief, the characteristic evacuation consists of blood and mucus closely intermingled with fluid offensive faeces, the blood occurring in streaks or clots, whereas in bacillary dysentery the stool consists of viscid, odourless, blood-stained mucus.

WILLMORE and SHEARMAN have pointed out that the cellular exudate of the "amoebic stool" has diagnostic characters that warrant a further extended search for the typical entamoebae in cases in which they were not found on the first examination. These may be summed up as follows:—

Scantiness of the cells, especially polymorphonuclears; preponderance of mononuclears over polymorphonuclears. Evidence of proteolytic digestion of the cells; absence of all phenomena characteristic of inflammatory reaction.

The microscopic diagnosis in the chronic or latent stage of amoebiasis is by no means so easily made. The appearance of the cysts in the faeces is so precarious that should but a single specimen of the faeces be examined a very considerable number of infections will be missed. This is especially true of the so-called cyst-carriers. DOBELL has recorded an instance in which a diagnosis was made on the thirty-fifth consecutive examination, and it is probably necessary as a routine that at least six stool examinations should be made. The position may be put this way:—Should a series of cases be examined once apiece, about one-third of the infections actually present may be disclosed. In a series thrice examined, between one half and two-thirds of the actual number of infections will have been detected.

The limitations of simple microscopic diagnosis have suggested methods of concentrating entamoebic cysts in stools as elaborated by CROPPER and ROW. The maximum concentration of cysts for diagnosis is obtained as follows:—Faeces and saline in the proportion of 1 gm. of faeces to 30 cc. saline are shaken on a shaking machine for a minimum of half an hour. The emulsion is then poured into a separating funnel and shaken up by hand for half a minute with 10 to 20 per cent. of its volume of ether and allowed to stand for a minute or two. The faecal matter absorbs the ether and rises to the top of the saline, while the cysts remain beneath; by this method a fifteen-fold cyst concentration is effected.

The identification of cysts is best effected by the addition of iodine solution to the stool; this brings out their salient diagnostic features. The solution is made by saturating a 1 per cent. watery solution of

potassium iodide with iodine, which acts partly as a fixative, partly as a stain. For still more accurate differentiation of cysts the following staining method is recommended by DOBELL. The specimen should be prepared and fixed as soon after the passage of the stool as is possible. The preparation should never be allowed to dry.

The following special reagents are required :—

1. *Fixative.*—Saturated aqueous solution of mercury perchloride, 2 parts ; absolute or 96 per cent. alcohol, 1 part. When mixed together 5 cc. glacial acetic acid should be added to each 100 cc.

2. *Stain.*—Haemalum solution, prepared as follows : one gm. of haemotoxylin is dissolved in 1 litre of water ; then 0·2 gm. of sodium iodate ($NaIO_3$) and 50 gm. of alum (potash) are added. When these salts are dissolved the stain is filtered. This stain has the advantages that it is easy of preparation, does not require to be kept before use, and is not liable to over-stain.

Fixation.—In preparing the specimen a small portion should be spread with a platinum loop on a cover-slip. As soon as the material is spread the cover-slip is floated filmside downwards on the fixative. Fixation is complete in 10–20 minutes.

Washing process.—The specimens must be now thoroughly washed, first in 50 per cent. or 70 per cent. alcohol, after which they are placed in 70 per cent. alcohol to which a few drops of iodine solution have been added. In this they are allowed to remain for 30 minutes, and are then placed in weaker alcohol for a few minutes before being transferred to distilled water.

Staining.—This is carried out by placing the cover-slips in the haemalum solution for 10 to 20 minutes. If over-staining occurs they may be decolorized in acid alcohol or weak alum solution. After staining the specimens are washed in tap-water until blue.

Dehydration, etc., is preferably carried out in stages—the specimens being placed first in 35 per cent., then in 70 per cent., and lastly in absolute alcohol ; from this they are transferred to a mixture of equal parts of absolute alcohol and xylol, then to pure xylol, and are then mounted in balsam. The greatest care must be taken that the specimen is never allowed to dry.

The association of Charcot-Leyden crystals with long-standing intestinal amoebiasis has been noted by ACTON, and more recently by THOMSON and ROBERTSON, who consider their presence in the stool as suggestive, if not diagnostic. These crystals are found also in scrapings of the ulcers, but their source is not clear ; they may be due to specific action on the part of the *Entamoeba histolytica*, as they are never found in dysenteries of bacillary origin.

In view of the fallacies and shortcomings of microscopic diagnosis MANSON-BAHR and GREGG advocate the routine use of the sigmoidoscope ; by this means the entamoebae may be demonstrated in scrapings of the ulcers obtained through the instrument. The characteristic appearances are oval or diamond-shaped ulcers varying greatly in size and having haemorrhagic margins and grey or greenish sloughs. Flame-shaped haemorrhages may also be visible, often partly concealed in folds of puckered mucous membrane.

REINHARD has claimed assistance in diagnosis from the employment of X-rays. The substance used is barium sulphate. Insufficiency and stenosis at the ileo-caecal valve and flexures may be thereby revealed and are said to be characteristic.

As regards the diagnosis of hepatic abscess CANDLER has recorded that X-rays do not always aid, should the abscess occur in the centre of the liver. Leucocytosis may be uncertain, as low as 4,700 and as

high as 28,000. GIBSON observes that a history of previous diarrhoea or dysentery is by no means conclusive; pain is necessarily present in every case of unilobar abscess owing to distension of the liver.*

6. *Treatment.*

Growing disbelief in the efficacy of hypodermic injections of emetine hydrochloride to effect a radical cure of amoebic dysentery is manifest in all the more important recent literature. WENYON and O'CONNOR, in Egypt, concluded that it was necessary to administer larger doses than had hitherto been considered judicious in order to effect a cure. They injected 1 gr. a day hypodermically, together with ½ gr. in keratin-coated tabloids by the mouth for 12 days, totalling 18 gr. This method was more efficacious in chronic carrier cases than in the acute stage of the disease. A further deterrent to the extended use of emetine is the fact that in excessive doses its toxic effect is distinctly cumulative. DALE produced definite poisonous effects in experimental animals, while KILGORE has described a peripheral neuritis after emetine treatment. BALFOUR and PYMAN have drawn attention to the fact that emetine may itself produce and keep up a certain degree of diarrhoea, and it is important to distinguish this drug-produced diarrhoea from that caused by intestinal disease.

Emetine bismuthous iodide in the treatment of amoebic dysentery was introduced by DUMEZ, and was quickly taken up by DALE, who suggested an average daily dose by the mouth of 3 gr. in capsules (representing 1 gr. of emetine hydrochloride), given on 12 consecutive nights—a total of 36 grains. The drug is a red powder insoluble in dilute acid, which, passing through the stomach unchanged, becomes gradually decomposed, with the liberation of emetine and precipitation of bismuth sulphide in the intestine.

It has been shown by LOW that the drug passes through the intestine unabsorbed if compressed into a hard tablet, or if coated with insoluble substances such as paraffin, vaseline, resin, keratin, or stearin. It is best made up in powder form in hard gelatin cachets. Vomiting and nausea may be prevented by 10–15 minims of tincture of opium given half an hour beforehand. Slight purging is the rule and should be regarded as a favourable symptom. During the course the patient must remain in bed and partake of a light fluid diet, preferably milk.

In chronic cases and cyst passers GUNN and SAVAGE and LILLIE and SHEPHEARD have reported that emetine bismuth iodide has given by far the most satisfactory results. Unfortunately it is now recognized that although 70–80 per cent. are cured by one course as described above, yet there remains a residue who are refractory. In these cases

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* REFERENCES.

Diagnosis.—NOC (F.), vol. 8, p. 112; WILLMORE (J. G.) & SHEARMAN (C. H.), vol. 12, p. 311; COPE (Z.), vol. 17, p. 35.

By X-rays.—REINHARD (P.), vol. 9, p. 177.

Detection of Cysts.—CROPPER (J. W.) & ROW (R. W. H.), vol. 9, p. 421; CARTER (H. F.) & MATTHEWS (J. R.), vol. 11, p. 46.

By Charcot-Leyden crystals.—ACTON (H. W.), vol. 14, p. 96; THOMSON (J. G.) & ROBERTSON (A.). *Proc. Roy. Soc. Med.* 1921, vol. 14, pp. 33–44.

By Sigmoidoscope.—MANSON-BAHR (P. H.) & GREGG (A. L.), vol. 18, p. 35.

Diagnosis of Hepatic Abscess.—GIBSON (C.), vol. 14, p. 316; CANDLER (A. L.), vol. 16, p. 2.

72 and even 200 gr. of EBI have been given altogether. If well tolerated it may be possible by splitting up the dose to give 4 or even 5 gr. a day.

Noc and others consider that emetine is rendered more efficacious by the intravenous injection of neosalvarsan, possibly by the tonic action of this drug. Ravaut and Krolunitzki have given novarsenobenzol in capsules, each containing 0.05 gm., daily by the mouth for 10 days in chronic cases and cyst passers.

Calame has reported rectal injections of neosalvarsan in doses ranging from 0.45 gm. in 200 cc. to 0.9 gm. in 1000 cc., and considers this method worthy of a further trial.

Nixon, Sellards and McIver, Shepheard and Lillie report favourably on treatment with "chaparro amargosa." This is a decoction made from the powdered roots, branches and foliage of a plant (*Castela Nicholsoni*) which is indigenous to Texas and north Mexico. Chaparro is closely allied to simaruba. Treatment should last ten days. Five teaspoonfuls of the powdered drug are used each day—three for oral and two for rectal use. The drink is prepared by boiling for a quarter of an hour one teaspoonful of powdered chaparro in 8 oz. of water; it is then allowed to cool and is strained. It is administered half an hour before breakfast, dinner and supper. The enemata, prepared in the same way as the drink, save that 12 oz. of water are used, are given one in the morning about 10 a.m., the other in the evening at 6 p.m. The patient should remain in the elbow-knee position for at least 15 minutes after the enema and should retain it as long as possible.

Dargein has advocated rectal lavage with infusions of ipecacuanha root, 10 gm. to 500 cc. of water.

Haughwout in China has obtained "surprisingly good results" with benzyl benzoate in 20 per cent. alcoholic solution, twenty drops being given three times a day.

Walker and Emrich have tried oil of chenopodium in 16 minim doses in cachets (given on three occasions at two-hourly intervals—making 48 minims in all) in cyst passers with good results; but on account of its poisonous action, chenopodium is not generally recommended.

Niklas states that appendicostomy with lavage has not proved to be a satisfactory method in chronic amoebic cases.

Emetine injections have proved to be a necessary adjuvant in the treatment of hepatic abscess. Dopter states that there is no evidence that this drug assists in the absorption of liver pus—evacuation is absolutely necessary; washing out the abscess cavity with a solution containing emetine is not recommended. Should the stools contain *E. histolytica* cysts a course of emetine bismuth iodide should be prescribed.*

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* References.

Treatment by Emetine.—Dale (H. H.), vol. 7, p. 237; Balfour (A.) & Pyman (F. L.), vol. 8, p. 219; Kilgore (A. R.), vol. 8, p. 442; Jepps (M. W.), vol. 9, p. 179; Wenyon (C. M.) & O'Connor (F. W.), vol. 11, p. 43.

Emetine Bismuthous Iodide.—Dumez (A. G.), vol. 6, p. 446; Dale (H. H.), vol. 8, p. 441 & vol. 10, p. 129; Low (G. C.) & Dobell (C.), vol. 8, p. 441; Dobell (C.), vol. 9, p. 178; Dale (H. H.) & Dobell (C.), vol. 11, p. 248; Savage (R. E.) & Young (J. R.), vol. 11, p. 49; Gunn (J. W. C.) & Savage (R. E.), vol. 15, p. 187.

7. *Prophylaxis.*

Efficient prophylaxis of amoebiasis depends upon an exact knowledge of the manner in which the infection is spread. Unfortunately this cannot be said to be satisfactorily established. It is generally believed that the infection is carried mainly by polluted water, though this belief cannot be said to rest upon any exact foundation. The danger of house-flies acting as carriers of infection has been emphasized by Wenyon and O'Connor in Egypt and by Buxton in Mesopotamia. The former observers have proved that the cysts of *E. histolytica* can be found in the fly's intestine so long as any faecal matter remains there. Should the flies be prevented from feeding, this interval may be as long as forty-two hours. The direct passage of practically unaltered material through the gut of the fly would seem to be of more importance in the distribution of the disease generally than the regurgitation of such material through the proboscis or its adherence to the legs or bodies of the insects.

The existence of large numbers of excretors of *E. histolytica* cysts has naturally a considerable bearing upon the prophylaxis of the disease. Carriers of entamoebae have been divided into two classes—generally termed *contact carriers* and *convalescent carriers*. The former are individuals who have never suffered from amoebic dysentery, and the latter those who have recovered clinically from dysentery without losing their infection. Highly infected individuals showing no clinical signs of infection and passing faeces which are infective to others probably greatly outnumber those who suffer from dysentery. Viewed in this light the occurrence of dysentery is an accident and is as much against the interests of the parasite as it is deleterious to the tissues of the host. According to Dobell, *E. histolytica*, whether it causes recognizable dysenteric symptoms or not, must always live at the expense of its host's tissues. The ulceration may be superficial or even microscopic.

Campbell, in Alexandria, performed autopsies on cases well known to be carriers without being able to find any naked-eye lesions, and we know also from the work of Bartlett that amoebic ulceration of the intestine may exist without producing dysenteric symptoms during life.

In order, then, effectively to protect a community from amoebiasis it would be necessary to detect and isolate all healthy carriers of infection. This manifestly cannot be done. The longer European troops

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Treatment of Carriers by Emetine Bismuthous Iodide.—Waddell (W.), Banks (C.), Watson (H.) & King (W. O. R.), vol. 11, p. 50; Lillie (D. G.) & Shepheard (S.), vol. 11, p. 50.

Alcresta Ipecacuanha.—Allan (W.), vol. 8, p. 113.

Salvarsan.—Noc (F.), vol. 8, p. 113; Ravaut (P.) & Krolunitzki (G.), vol. 8, p. 442; Calame (P.), vol. 12, p. 8.

Oil of Chenopodium.—Walker (E. L.) & Emrich (W.), vol. 11, p. 51.

Benzyl Benzoate. — Haughwout (F. G.) & Lantin (P. T.), vol. 15, p. 190; Haughwout (F. G.), vol. 17, p. 37.

Chaparro Amargosa.—Nixon (P. I.), vol. 6, p. 51, and vol. 8, p. 114; Shepheard (S.) & Lillie (D. G.), vol. 12, p. 7; Sellards (A. W.) & McIver (M. A.), vol. 13, p. 225.

Ipecacuanha Enemata.—Dargein (G.), vol. 17, p. 163.

Surgical Treatment.—Niklas (F.), vol. 17, p. 36.

Treatment of Hepatic Abscess.—Dopter (Ch.), vol. 8, p. 114; Gaglio (E.), vol. 17, p. 166.

remained in the endemic areas of amoebiasis, the higher the percentage of healthy excretors became. After a year's residence in Egypt WENYON and O'CONNOR found that the percentage of carriers amongst the troops was about 5·3 per cent., whilst it was much higher—13·7 per cent.—amongst native Egyptians. At the end of the Mesopotamian campaign MACADAM, on examining the British troops returning to India, estimated that no less than 33 per cent. were carriers of *E. histolytica*.

WENYON and O'CONNOR, in considering this problem, remarked that, even if the organization for the examination of very large numbers of troops existed, it would be unjustifiable to detain large numbers of healthy men, for supposing that all carriers amongst the troops were separated and treated, there yet remains a source of infection in the shape of the native of the country and the house-fly, against which scientific armature is at present helpless.

The prophylaxis of amoebiasis, as in the case of other familiar intestinal diseases, may be said to rest upon an efficient general sanitation.*

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* REFERENCES.

Carriage by Flies.—WENYON (C. M.) & O'CONNOR (F. W.), vol. 10, p. 126; BUXTON (P. A.), vol. 16, p. 1.

Cyst passers.—MACADAM (W.) & KEELAN (R.), vol. 16, p. 2; MACADAM (W.), vol. 17, p. 1; LEDINGHAM (J. C. G.), vol. 17, p. 31.

MALARIA.

i. Christophers (S. R.) & Shortt (H. E.). **Malaria in Mesopotamia.**—*Indian Jl. Med. Res.* 1921. Jan. Vol. 8. No. 3. pp. 508–552. With 4 maps, 1 chart and 2 plates.

ii. —— & ——. **Incidence of Malaria among Troops in Mesopotamia, 1916–1919.**—*Ibid.* pp. 553–570. With 1 chart.

These papers form a valuable contribution to our knowledge of malaria as it affects the Mesopotamian plain and submontane regions, and the montane regions of Persia between the 30th [not 35th] and 37th parallels. The level alluvial riverine plain, sterile apart from irrigation, passes westward into the practically waterless, undulating, stony or sandy desert, and eastwards into submontane and mountainous country. Irrigation is (i) as in the Punjab, by high-level canals, with attendant risk of spilling and waterlogging; (ii) by uplift, in which case water is expensive and but little is allowed to run to waste; (iii) by inundation in the lower reaches where, as the result of alluvial deposit, the river banks overtop the surrounding country, and where, by merely breaching these, water may be obtained in unlimited quantity; with the result that such overflow runs freely on to the land, whose subsoil water is thereby raised in surface level, a fact having a most essential effect on prophylaxis about Basra; (iv) " palm grove irrigation " in the Shat-el-Arab, consisting of a series of branching creeks bringing tidal but fresh water within a few feet of every part of a belt three to seven miles wide.

The Anopheles of this region are:—(1) *A. pulcherrimus*, very common and almost ubiquitous in the plains from March to November, a strong flier, marsh breeder, voracious, mainly a night feeder, bold and unretiring, rarely, if ever, associated with marked malarial prevalence, but proved capable of acting as host. Its area of distribution in Mesopotamia is eastwards from the Syrian desert along the Tigris plain. It reaches India by the Oxus and Indus plains.

(2) *A. superpictus*, a breeder, in Mesopotamia, in submontane river pools and running water and in irrigation channels, retiring, always associated with a high malarial incidence, a proved host of malarial parasites, ranging from Abbottabad and Quetta to Salonica, Greece, Southern Italy and Algeria.

(3) *A. maculipennis*, the chief European malaria carrier, has in Mesopotamia a distribution corresponding to that of *A. superpictus*, and breeds there during the cold weather (the larva and pupa surviving freezing) in irrigation channels and irrigation leaks. It has no evident association with malaria.

(4) *A. sinensis* is an undomesticated cold-weather breeder in swamps and irrigation channels, probably with little relationship to malaria in Mesopotamia.

Other species occasionally found there are *A. algeriensis*, *A. bifurcatus* and *A. rhodesiensis*.

As indicated by the spleen rate in 7,981 children, there is moderate malarial endemicity about the Shat-el-Arab (spleen rate 12) and in the lower Tigris and Euphrates (28); an area of slight endemicity in the upper Tigris and Euphrates (3·9), extending into the Dyala submontane; hyperendemicity in the Zab (66) and Mosul (77) submontanes, and a hyperendemic focus at Kerbala (81); while the Persian plateau and Bagdad and Mosul cities are free from infection.

That infection and admission to hospital were not synonymous among the 400,000 troops attached to the Mesopotamian Force is shown by the fact that blood examinations of 3,500 men doing active duty with their units gave a percentage of infection for British troops of 1 and for Indian troops of 2·7. This implies that on any one day 1,000 British and over 8,000 Indians were infected, while only 27 and 31 respectively sought admission to hospital, an important aspect of the subject requiring further elucidation. Quartan malaria caused not more than 1 per cent. of the cases; the others were predominantly benign tertian, though less markedly so in the northern areas.

Clayton Lane.

Wenyon (C. M.), Anderson (A. G.), McLay (K.), Hele (T. S.) & Waterston (J.). **Malaria in Macedonia, 1915-1919. Part I. The Incidence and Aetiology of Malaria in Macedonia** [by Wenyon (C. M.)].—*Jl. Roy. Army Med. Corps.* 1921. Aug. Vol. 37. No. 2. pp. 81–108. [First instalment.] With 1 map, 1 chart and 9 illustrations.

British troops occupied nearly 2,000 square miles of Macedonia, the district consisting of a fringe of valley area haunted by *Anopheles maculipennis*, and a hilly centre swarming with *A. superpictus*, the whole, however, being highly malarious. Time, men and money were none of them available to rid this large area of mosquitoes. An enormous amount of anti-mosquito work was indeed done, but its magnitude necessitated its limitation within a circumference of half a mile from camps. Since local evidence became forthcoming that the range of flight of mosquitoes was not a quarter of a mile, as had been supposed, but one, two, or indeed anything up to ten miles with a favouring wind, the extent of this work, great though it was, proved quite ineffective in making any appreciable diminution either in the numbers of mosquitoes or in the incidence of malaria. As is aptly said, "When the number of mosquitoes attacking an individual amounts to hundreds, if not thousands, the reduction of these by a small proportion will hardly lessen a man's chance of infection. It matters little whether he is bitten by ten or only by five infected mosquitoes in a single night."

Quinine prophylaxis in Macedonia proved most disappointing. Wenyon's view on the subject is this. If one may assume that, as in trypanosomiasis, there are certain individual parasites insusceptible to the curative drug, so in malaria there are certain sporozoits, or young forms arising immediately from them, which are resistant to quinine, it will follow that "if an individual is being bitten by only a small number of infected mosquitoes the chances of his acquiring malaria will be very much diminished if he has quinine in his system, for it will be only very rarely that resistant sporozoits will be injected. If, however, he is being constantly bitten, he will most certainly be infected, for among the large number of sporozoits injected there will be some resistant forms." That the percentage of infected mosquitoes must have been great was shown by the finding of 25 per cent. of carriers in about 3,000 men who had suffered clinically, and by the fact that anything up to 70 per cent. of the mosquitoes found in tents in the morning had fed during the night. Mosquito nets used were of two types: a bivouac net taking two men, and a bell net fixed to a hoop.

hung over the head of each person. The disadvantage of the former was that it had to cover two men, of the latter that it was difficult to keep adjusted, of both that they were stores issued locally after men reached a camp, and that there was frequently a delay of one, sometimes of two, nights before such issue was made to newly arriving units. It is suggested that the issue to rest camps of single rectangular nets, with a calico base, in numbers in excess of strength to allow for repairs, and arranged on cords in parallel rows, will afford the best protection. Screened huts proved valuable, as did minor devices such as turn-down shorts to protect the knees, sprayers, swats, traps, and repellents, the last useless half an hour after application. The conclusion reached is that in this highly malarious country, had the money and energy expended in antilarval measures and in quininization been concentrated on the protection of man against actual bites, malaria would probably have been lessened.

Wenyon next considers the published reports of malaria in the French zone, lying immediately to the west of that held by the British. Although mosquito netting, mosquito proofing and antilarval measures were carried out on a less lavish scale than by the British, and although the latter had more malaria in 1917 than in 1916, it is claimed that for every 60 cases of primary malaria in 1916 the French had only 7 in 1917. On analysing available figures it appears, however, that in 1916 they had 31,727 admissions to hospitals for malaria. Of these, 17,614 were evacuated to France and 13,446 left in Macedonia. Yet in 1917, 31,000 cases of secondary malaria were admitted to hospital. Presumably primary were differentiated from secondary attacks on the statement of the individual soldier. This same procedure, at one time adopted in our own army, was abandoned on account of its unreliability.

The Serbians, too, suffered badly from malaria, as also did the Bulgarians, a fact learned after the Armistice.

C. L.

HAMILTON (C. S. P.). **Malaria : Two Years' Experience on the Salonika Front.**—*Jl. Roy. Army Med. Corps.* 1921. May. Vol. 36. No. 5. pp. 361–369. With 1 chart in text.

One of the points on which stress is laid in this picturesque narrative is the influence attributed to lowered vitality in preventing the effective treatment of malaria by quinine ; the lowering of vitality being itself assigned to heavy work and a life of unrelieved monotony, and passing by the local name of " Balkan tap." It had apparently been expected that mosquitoes would not be found at a height of over 2,000 feet, although experience in India alone would negative this, while their maximum flight was put at about 2,000 yards. [See, however, WENYON'S article on Macedonian malaria.]

Instances are quoted of benign tertian fever with most indefinite symptoms, the diagnosis being made by blood examination. The prophylactic measures referred to were the anointing of exposed parts with Para Quit ointment, the issue of masks, gloves and bivouac mosquito nets, and quinine. In spite of them a 60 per cent. infection existed among the troops at one time. The treatment which is believed to have been generally carried out consisted in the administration of 40 grains of quinine in the first 24 hours, 30 grains in the second and

40 grains in the third, after which quinine was replaced by iron and arsenic. That this inadequate dosage was only part of the reason for the disrepute into which quinine fell locally is evident from the perusal of WENYON's paper, to which reference has already been made.]

C. L.

KEUKENSCHRIJVER (N. C.). **Eenige waarnemingen over Malaria bij de Hollandsch-Amerikaansche plantage mij. te Kisaran.** [Some Observations on Malaria in the Holland-America Plantation Company at Kisaran.] — *Geneesk. Tijdschr. v. Nederl.-Indië.* 1921. Vol. 61. No. 2. pp. 202–210. With 1 double plate.

This paper contains in a concise form much information about a malarial epidemic at Kisaran (East Coast Sumatra). Several (10) species of Anopheles were found on the rubber estates in question, of which *A. kochii* is considered to be the principal carrier. It was found infected in nature, and could be infected experimentally with benign tertian and malign subtertian in 5–10 per cent.

Often malarial parasites were found in the blood of patients without enlargement of the spleen. This is ascribed to the great quantities of quinine distributed on the estates.

Treatment of all parasite carriers was not sufficient to combat the epidemic, neither was treatment of all people with enlarged spleen, either with or without parasites in the blood. To reduce the chances of infection both groups should be treated daily for a considerable time with quinine.

Other accessory measures are valuable (improved feeding, drainage, oiling, light work, mosquito netting). Importation of new coolies proved to have a bad influence on the spread of the epidemic.

W. J. Bais.

GILL (C. A.). **The Rôle of Meteorology in Malaria.**— *Indian Jl. Med. Res.* 1921. Apr. Vol. 8. No. 4. pp. 633–693. With 1 chart, 1 map and 1 diagram.

Malaria is here considered as manifesting itself in two different ways: (*a*) epidemic malaria, evidenced by "determining, in the case of each district, the 'coefficient of variability' of 'fever' mortality, the calculation being made by multiplying the standard deviation of the autumnal 'fever' mortality (October and November deaths) by 100 and dividing the product by the mean 'fever' deaths during the same period"; and (*b*) endemic malaria, evidenced, provided no epidemic outburst has occurred within the last five or six years, by the spleen rate in children.

It is the case in the Punjab, with which alone this paper deals, that those areas with a high rate of endemic malaria are not those in which epidemic outbreaks occur. There is, moreover, a close association between epidemic areas and areas liable to flooding. As to the cause of this difference, there is epidemiological evidence that malaria in the Punjab is only transmitted if the mean monthly humidity (8 a.m. reading) be at least 63 per cent. and the mean monthly temperature at least 61° F. In support of the statement that it is precisely in those areas where these minima are least frequently attained that epidemic malaria is rifest there are cited two facts: (1) there is a close negative correlation between the intensity of epidemic malaria

and the mean annual potential infection period as measured on the criteria just mentioned; (2) in the Montgomery district epidemics are rare, yet when they occur they are of an intensity unequalled elsewhere in the Province. During the 17 years over which meteorological records are available there have been two periods of 30 months each during which there has been complete cessation of potential infection. Interruption of infection is accordingly looked upon as an essential factor in the mechanism of epidemic malaria.

On the basis of these general epidemiological findings experimental work was undertaken to test the effects of temperature, humidity and low atmospherical pressure (that is, inferentially, of altitude), on mosquitoes, particularly on their length of life, habits and power to transmit infection; and since in Lahore in April and May suitable malarial cases and Anopheles are both difficult to obtain, the experiments actually dealt with the parallel Proteosoma infection in *Culex fatigans* and sparrows. It was found that with a relative humidity of less than 40 per cent. Culex would not feed; with one between 40 and 48 per cent. Culex fed, but if infected died within five days; while with one over 50 per cent. they fed freely and survived. Accordingly Proteosoma cannot be transmitted with a humidity of less than 48 per cent. Regarding temperature, the details which apply to Culex and Proteosoma are not evidential of Anopheles and Plasmodium. For example, at 40° C. *C. fatigans* dies within 15 minutes, whereas *Anopheles culifaciens, A. stephensi* and *A. pulcherrimus* remain full of life. Jansco and others have shown that the lower limit of growth for *P. vivax* and *P. falciparum* in Anophelines is 16° C. (60·8° F.). As regards atmospheric pressure it was shown that between 25° and 28° C. with a relative humidity approaching 100 per cent. and with the mercury standing at 23 inches, corresponding to an altitude of between 5,000 and 6,000 feet, the development of *P. grassii* in *Culex fatigans* proceeds normally. Clearly, it is concluded, the rôle of meteorology in the transmission of malaria is important and claims definite medical recognition.

C. L.

Roubaud (E.) & Leger (M.). **Observations sur le paludisme en Corse (mars-avril 1921).**—*Bull. Soc. Path. Exot.* 1921. June 8. Vol. 14. No. 6. pp. 340–351.

The work here recorded represents a rapid survey, made under the auspices of the Pasteur Institute at the end of March and beginning of April, 1921, of the general conditions prevailing in Corsica, with a view to the re-institution of antimalarial measures, which had been dropped during the war.

From the examination of 37 children, of which 16 were found infected, and from the comparison of these results with those previously obtained by Leger, it is concluded that the belief that within the last seven years malaria in Corsica has greatly increased is correct. The increase is attributed to conditions brought about by the war. Of 20 *Anopheles* [*maculipennis*] examined, young sporocysts were found in one—the salivary glands being free from infection in all. Of the 16 positive blood examinations, two contained gametes of *Plasmodium vivax* and one of those of *P. praecox*. These findings are in direct contradiction to those of Grassi, who finds crescents absent at Fiumicino, near Rome, till the middle of July, the difference being explained by the fact that the Corsican examinations were made at

random and without any reference to history or clinical signs. They are taken to show that the possibilities of infection by the two parasites are at that period of the year practically identical, the priority of new infections in mass by the benign tertian parasite being attributed to its more rapid development in the mosquito, a physiological explanation which appears to the writers to fit better with the facts than does the assumption, with no proof offered, of a polymorphism.

Reverting to Roubaud's belief in the efficiency of animal houses as preventives of human malaria, it is pointed out that the claim has never been made that any type of stable, byre or hutch will serve this purpose effectively. To do so it must be of a height not exceeding 3·5 metres, sheltered from wind and sun, and animals must be regularly stabled therein at night; the intention being that the mosquitoes, having found happy conditions for food and housing within a few feet of one another, will rest therewith content. In such a byre, and only one such was found, many mosquitoes hung replete, the neighbouring dwelling-house being empty of them. Such animal houses, properly placed, built and used, can, it is believed, be made effective screens to keep human beings free from Anopheline attack.

Regarding breeding places it is pointed out that the large lagoons communicating with the sea are not, as is generally supposed, breeding places for Anopheles, although the canals which form the communications are so.

C. L.

Regnault (Félix). **Les causes adjuvantes du paludisme.**—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1921. June 23. Vol. 45. No. 21. pp. 927–929.

This paper deals actually with the conditions which favour the spread of malaria in Corsica. They are defined as overcrowding within the house, the whole family sleeping in one room, with the result that when one becomes infected the others establish a sort of monopolistic trust in the mosquitoes which feed on the infected member, the insects reciprocating by infecting them all; site crowding of houses, the better to avoid the effects of the vendetta; absence of animal houses, domestic beasts being free-living; poverty; ignorance manifesting itself as a belief that the mosquito is no carrier, but that the trouble arises from bad air. Education of children is suggested as the only way out of the present difficulties.

C. L.

Ferguson (Eustace W.); Hughes (Laurence H.); Evans (Wilfred). **The Malaria Danger.** [Correspondence.]—*Med. Jl. Australia.* 1921. 8th year. Vol. 1. May 21. No. 21. pp. 432–433.

In the correspondence which has naturally followed upon the publication of autochthonous cases of malaria in New South Wales [this *Bulletin*, Vol. 18, p. 89], Ferguson, writing from the office of the Director-General of Public Health of New South Wales, points out that the following Anophelines have so far been found in Australia.—*Anopheles corethroides* and *A.* (*Myzorhynchus*) *barbirostris bancrofti*, but not from New South Wales; *A.* (*Pyretophorus*) *atratipes*, but not abundantly; *A.* (*Pyretophorus*) *stigmaticus*, once only; *A.* (*Nyssorhynchus*) *annulipes*, the common Australian Anopheline, extending

from Tasmania to the islands on the north. The possibility of the occurrence of endemic malaria has received serious consideration, and it has been concluded that such is unlikely to occur, since Anophelines are relatively scanty in urban, and human beings in rural, areas. The Murrumbidgee irrigation area is an exception, and here the policy has been to exclude settlers who are known to have suffered from the disease.

Wilfred Evans draws attention to a case of malaria reported by him in 1919 [this *Bulletin*, Vol. 15, p. 422], and points out that the grave apprehensions of the likelihood of a serious Australian epidemic, which he then voiced, have not been justified.

C. L.

Doyle (Gerald). **Clinical Reports of Two Unusual Cases.** [One Malaria.]—*Med. Jl. Australia.* 1921. 8th year. Vol. 1. May 21. No. 21. p. 421.

A man who had never been out of the State of Victoria, and had spent all his life at St. Arnaud, with the exception of a fortnight at Mildura, latitude about 34·2° S., developed typical tertian malaria, confirmed by the finding of *Plasmodium vivax*.

C. L.

Grassi (B.). **L'Anofele può propagare la malaria anche direttamente?** [Can Anopheles transmit Malaria directly?]—Reprinted from *Reale Accademia Nazionale dei Lincei.* 1921. Jan. 2. Vol. 30. No. 1. p. 2.

In the summer of 1920 Anopheles were comparatively scarce in Fiumicino, and of 200 examined all were found to be uninfected. Moreover, cases of relapse in chronic malaria patients were very few.

In a cottage in this district, where no relapse had occurred, a baby, a few months old, developed a primary subtertian infection. A youth significantly infected with the same form of parasite had stayed in the house about a week previously.

From this and other similar local coincidences Grassi wonders whether malaria may be transmitted directly by a mosquito regurgitating infected blood into the body of a healthy individual.

It should be quite possible to test this theory experimentally.

J. Rosslyn Earp.

Schlesinger (W.). [**Activation of Chronic Malaria.**]—*Wien. Arch. f. innere Med.* 1921. June 30. Vol. 2. No. 3. p. 241. [Summarized in *Jl. Amer. Med. Assoc.* 1921. Aug. 27. Vol. 77. No. 9. p. 742.]

Believing that the appearance of parasites in the blood stimulates production of antibodies and is therefore a favourable sign, and aiming at inducing such appearance without consequent symptoms, Schlesinger suggests that the problem may be solved, not by intragluteal injections of 2–3 cc. of a 10 per cent. solution of sodium nucleinate, as he himself has done, but by the rectal administration of nucleinic acid in the form of active yeast, or by the oral administration of calf's thymus or other food rich in this substance. He suggests a correlation between the flaring up of malaria in the spring and the eating of fresh green vegetables. So far as one can gather from the abstract the matter is put forward as pure conjecture.

C. L.

Labastie. **Des rapports du paludisme avec certaines infections.**—*Presse Méd.* 1921. Aug. 24. No. 68. pp. 1221–1222.

Noting that among troops in Cilicia those heavily infected with malaria were not found showing lesions of syphilis or tuberculosis, that in 23 cases with tubercular lesions only four, or 17 per cent., suffered from malaria, the type being benign in all instances, while in 10 cases of syphilis, only one, or 10 per cent., showed a benign tertian infection, and that only after the administration of neosalvarsan, while the general percentage of malarial infection among the troops was 63, the writer concludes that there is an antagonism between malaria and these two other diseases. He attributes this immunity against malaria to the hyperleucocytosis induced by tuberculosis and syphilis, and finds confirmation of his belief in that his figures show special rareness of malarial infection when there exist glandular and osseous tubercular lesions—lesions, that is, of the tissues which produce leucocytes.

[The argument implies that tubercular inflammation induces excessive action of affected organs and tissues, but since the diagnosis is throughout purely clinical it is unnecessary to follow the writer further.]

C. L.

Blacklock (B.). **Notes on a Case of Indigenous Infection with *P. falciparum*.**—*Ann. Trop. Med. & Parasit.* 1921. Apr. 27. Vol. 15. No. 1. pp. 59–72. With 2 text figs.

Blacklock reports further on the recent fatal indigenous case of malaria reported from Liverpool [this *Bulletin*, Vol. 17, p. 136]. In the blood after death one third of the erythrocytes contained young trophozoits, many with two, three, four or five parasites, and there were present in nearly equal, but in small numbers crescents, and fully segmenting forms each with 10 to 24 merozoits. Nucleated erythrocytes numbered 4·9 per cent. of the total leucocytes, and pigment was present in the mononuclears and in the plasma. The spleen contained a few spherical, oval, or crescentic gametocytes, fifteen times as many segmenting forms with 8 to 22 merozoits, and considerable black and golden pigment. In the bone marrow the picture was quite different. There were a few segmenting forms, ten times as many crescentic and oval gametocytes, and many eosinophilic large mononuclears.

It is concluded that in all probability the fatal attack was a primary one; that, there being nothing to support Ross's query as to the possibility of direct inoculation of infected blood, the vector was an infected mosquito; and that, should the infection, as previously suggested, have been obtained at a neighbouring health resort, this would leave seven days for parasitic incubation, four days during which sporulating parasites were present in the blood, and five more, for the formation of crescents—all reasonable periods.

Stephens and Christophers' suggestion that the delayed appearance of crescents, compared with that of merozoits, is due to the lapse of time required for the appearance of immunity is not accepted, it being pointed out that many primary cases in which the process of crescent formation is already established in the bone marrow nevertheless die; and the late appearance of crescents in the peripheral blood "is explained on the ground that the source from which they arise," namely, the bone marrow, "is limited in extent."

C. L.

CODA (Maria). **Anemia perniciosa e malaria.**—*Policlinico.* Sez. Med. 1921. Mar. 1. Vol. 28. No. 3. pp. 123–136.

Two cases which have a bearing on the relation of malaria to pernicious anaemia are described in this paper.

P.B., a soldier of the class 1878, developed malaria on active service in 1916, though this was not the first occasion on which he had been infected. He recovered sufficiently to resume his trade as a carpenter, and then came back for treatment on account of anaemia. The red cell count when first taken was 900,000, and subsequently fell as low as 650,000. He had anorexia and diarrhoea. Achylia was noted. The blood picture was typical of pernicious anaemia : the colour index was high, 1·1 to 1·5 ; there were present anicytosis, poikilocytosis, hyperchromic megalocytosis, normoblasts and megaloblasts, leucopenia, with relative lymphocytosis.

The patient was treated with Baccelli's mixture. He developed first an irregular fever and then definite malarial attacks. Large doses of quinine were added to the treatment, and the malaria was controlled. Under continued treatment with Baccelli's mixture the blood count also began to improve. He left hospital with a red cell count of 3,500,000, a white cell count of 6,000, and a colour index of 1. He has since been under observation for four years, and at present is well, with a perfectly normal blood picture.

The second case, also a carpenter by trade, was found with severe anaemia at the beginning of what he believed to be his first attack of malaria. On the first day the red cell count was 1,200,000, with some anicytosis, anisochromia, especially hyperchromia, a few normoblasts and myelocytes, and very few megaloblasts. The percentage of haemoglobin was 35 and the colour index 1·4. Parasites were found in the blood from the same day. The anaemia improved rapidly, however, under quinine treatment. The rapid recovery in this case might lead to some doubt as to whether it was really of the type first described by ADDISON, though the blood picture is typical enough.

J. Rosslyn Earp.

CASTELLANI (Aldo) & WILLMORE (J. Graham). **Glycosuria of Malarial Origin.**—*Brit. Med. Jl.* 1921. Aug. 20. p. 286.

In the first of the two cases here detailed a man of 32, pale and tremulous, with no physical signs, with a blood showing a normal count but no malarial parasites, with sugar up to 8·5 gm. per litre of urine, and a history of a first attack of malignant tertian malaria about four years earlier, followed by numerous subsequent relapses, was given three 10-gr. doses of quinine daily and later an additional course of six intramuscular injections of quinine hydrochloride of 6 gr. each. The glycosuria disappeared, although no alteration had been made in the diet.

In the second case a man with polyuria, increased hunger and thirst and progressing emaciation, with a urine in which sugar had reached 2 per cent., very anaemic and earthy-looking and showing patches of hyper-pigmentation, with a spleen just palpable and very hard, with no malarial parasites in the peripheral blood, but a history of severe malarial infection contracted some four years earlier in the Balkans, was placed on a very strict diet by a medical man before coming to the writers. This led to but little reduction in the glycosuria. After taking 10 gr. of quinine thrice daily for three weeks the amount of sugar decreased to 0·1 per cent. Believing that there was here an instance of a diabetic syndrome in a malarial subject, he was permitted to revert to an ordinary diet. The glycosuria did not increase. While

in South Europe on business the patient stopped taking quinine, and was rewarded on his return by a rigor accompanied by high fever and sweating, the spleen becoming hard and palpable, a few malarial rings appearing in the blood, and the glycosuria reaching 1·2 per cent. He was placed on an intensive quinine treatment by mouth, supplemented by intramuscular injection, for six weeks, the diet not being restricted. Not only did the evidence of the chronic malarial infection disappear, but the urine became completely free from glucose. There is, then, reason for believing that glycosuria may be of malarial origin.

C. L.

Harrison (G. A.). **Glycosuria of Malarial Origin.**—*Brit. Med. Jl.* 1921. Oct. 22. pp. 630–631. With 1 chart in text.

Referring to Castellani & Willmore's case, it is reported that a Jew, aged 32, contracted malaria in Indore (India) in 1920, and next year developed excessive thirst, with glycosuria. In England the malaria recurred, but on a full diet the sugar found did not exceed 0·2 per cent. He was treated with full doses of quinine, after which 50 gm. of glucose in the morning on an empty stomach failed to produce glycosuria. Speculation as to the mechanism of production of the glycosuria follows.

C. L.

Fróes (Joãs A. G.). **A Case of Afebrile Quartan Malaria, with Urticaria.**—*Jl. Trop. Med. & Hyg.* 1921. Aug. 15. Vol. 24. No. 16. pp. 215–216.

A coloured woman of 25, resident in a malarious district in Brazil had never had clinical malaria, but came under observation as suffering every fourth day at eleven o'clock from urticaria. A fortnight's observation confirmed the statement but revealed no physical signs; it disclosed, however, quartan parasites in the blood, as had been suspected. Clinical cure, without disinfestation, followed the administration of quinine. The writer has had two similar cases caused by *Plasmodium vivax*, and notes that he has observed malarial polyneuritis.

C. L.

Leighton (William E.). **Spontaneous Rupture of the Malarial Spleen, with Abstract of Cases reported between 1842 and 1921.**—*Annals of Surgery.* 1921. July. Vol. 73. No. 1. pp. 13–19.

This paper narrates the case of a white waiter, aged 35, admitted to hospital with a two weeks' history of malaria, "malarial plasmodia of the tertian variety" in the blood, a rigid abdomen and epigastric and left hypochondriac tenderness, a tender, enlarged spleen, a temperature running to 102°, and an appearance as of shock. Severe abdominal pain supervened at night, and a ruptured spleen was diagnosed. At operation two parallel rents were found on its outer surface, and since tampons controlled the bleeding it was not excised. Bronchitis and cough developing, the wound, closed only by plaster, opened, and the omentum protruded, necessitating the removal of the tampon and the sewing up of the wound in layers, with drainage. Recovery followed. The accompanying abstract of cases will be found most useful, provided it is not assumed to be complete.

C. L.

i. Acton (Hugh W.), Rennie (P. M.), Curjel (Dagmar F.), & Dewey (J. O.). **The Diagnosis and Treatment of Benign Tertian and Malignant Tertian Fevers. Sections I–III** [by Acton, Curjel & Dewey]. **Section IV.—What Constitutes a Cure** [by Acton]. **Section V.—The Effect of Quinine on Benign Tertian Infection** [by Rennie, Acton, Curjel & Dewey]. **Section VI.—The Effect of Repeated Courses of Quinine on the Benign and Malignant Tertian Parasites** [by Acton, Curjel & Dewey]. **Section VII.—The Curative Value of the Total Alkaloids (Cinchona febrifuge) of Cinchona Bark on Benign Tertian Infections** [by Acton, Curjel & Dewey].— *Indian Jl. Med. Res.* 1921. Apr. Vol. 8. No. 4. pp. 750–773. With 3 coloured plates : 774–786. With 1 folding chart : 787–852 : 853–860 : 861–871.

ii. Acton (Hugh W.). **The Value of Quinine Prophylaxis.**— *Ibid.* pp. 741–749.

i. Situated at Dagshai, in the sub-Himalayas, at an altitude of 6,000 ft., where anopheles are rare and where in a four years' experience Acton has never seen a case of malaria which could reasonably be classed as having been locally contracted, a depot for soldiers, proved microscopically to have been infected with one of the malarial parasites, afforded the material on which this work is based. All these men had been given quinine before reaching Dagshai. After their arrival their outstanding feature was the almost regular occurrence of relapses due to the benign tertian parasite, and the almost complete absence of those due to the malignant tertian one. The conclusion reached is that quinine is a specific poison for the latter parasite, but not for the former. To establish, or facilitate criticism of the writers' diagnosis the trophozoits of the three forms of malarial parasite are distinguished by three coloured plates, full written descriptions, and a table, here reproduced.

Benign Tertian.	Malignant Tertian.	Quartan.
Accolée forms :—		
1. Usually lateral in position.	1. Lateral in position.	1. Central in position.
2. The parasite is continuous with the contour of the erythrocyte.	2. Projecting from the erythrocyte like a nipple.	2. Bird's eye in appearance.
	3. Rarely anaplasma forms seen.	
Pseudopodial forms :—		
1. Irregular kite - like forms with reniform or bipartite chromatin.	1. Bizarre in shape, usually a single pennant with division of the chromatin.	1. When viewed on the flat, comet or flame shaped.
2. The erythrocytes are enlarged, with Schüffner's dots.	2. Some of these may simulate an equatorial band form.	2. When viewed on the side, as an equatorial band form.
3. Rarely dendritic forms seen.		
Non-pseudopodial forms :—		
1. Large rings or globular forms in enlarged erythrocytes with Schüffner's dots present.	1. Small rings with early fission of chromatin, or signet ring-like, in corpuscles that are slightly smaller than normal.	1. Parasite seen elliptical in shape and as it grows larger assumes the equatorial band form. Corpuscle not enlarged.

Judged by these standards malignant tertian infections were almost absent in the cases under consideration. That this absence was not due to a previous faulty diagnosis having confused the young trophozoits of the two tertian forms (quartan forms were almost entirely absent) was proved by the comparison of 102 cases in which crescents, a form practically unmistakable, had been recorded in the medical history sheets, with 102 cases in which the history sheet recorded a microscopic diagnosis of benign tertian infection. In the first series there occurred during eight weeks of observation 64 benign tertian, and in the second 76 benign tertian, relapses, no malignant tertian relapses having occurred in either series. That this state of affairs was not due to a seasonal prevalence was shown by its persistence throughout the autumn and winter months, for in those months the prevalent infection in these areas is the malignant tertian. It is accordingly concluded that malignant tertian infection must have been eliminated by quinine treatment before the cases reached Dagshai. That being granted, to what is this elimination due? Is the benign tertian a mutation form of the malignant tertian parasite produced either spontaneously or, as the French school holds, as the result of quinine administration? Against this are the facts that the two forms differ in morphology, cultural characteristics, incubation period and symptomatology, to which is now added on the basis of this work, reaction to quinine, and by the fact that benign tertian malaria appears where no quinine has been taken. It is accordingly concluded that the benign tertian parasite is quinine-resistant.

The point next considered is the extent to which quinine will cure benign tertian fever, and as a preliminary it becomes necessary to establish the sense in which the term " cure " is used. The mechanisms of natural and artificial cures are clearly looked upon as quite different. It is evidently not considered the case that quinine aids the natural processes of cure. Natural cure is stated definitely to result from the gradual conversion of the whole of the asexual cycle into gametocytes, which subsequently, and shortly, die of senility. The validity of this contention is apparently dealt with in section VIII, not yet published. On the other hand, quinine and other cinchona alkaloids are considered to cure by destruction of the asexual cycle, an eventuality which, it must be concluded, is not considered to occur naturally.

Cure occurs in three phases (i) immediate, relative [or clinical] cure and (ii) partial cure, both effected by destruction of the greater part of the asexual cycle, there being in the latter case reappearance of parasites in sufficient numbers to cause symptoms; and (iii) complete cure, the result of complete disinfestation.

Cure brought about by quinine is looked upon as a fractional process, a certain and definite proportion of each new cycle being destroyed by each course of treatment. Provided sufficient has been given and absorbed, the efficacy of quinine is considered as dependent upon the number of parasites present and the rate of their destruction, while its failure is explained as a question of relative accessibility. The argument runs thus. Ramsden and others have shown that 90 per cent. of quinine injected intravenously disappears from the blood within one minute, accumulating in the tissues. Benign tertian parasites multiply in the blood, where the concentration of quinine is slight and interferes little with schizogony and the growth of young forms. Malignant tertian parasites multiply in the organs

where quinine is concentrated and so are readily destroyed. The argument seems to assume that concentration of quinine is greater in blood passing through quinine-containing tissues, or alternatively that schizogony may occur in those tissues and not in the blood.

Having established the senses in which the term cure may be used, a return is made to the effect of quinine on benign tertian infections. It was found that in 100 cases 2 to 5 intravenous injections of the acid hydrobromide or the bihydrochloride of quinine in doses of 5 to 10 gr., followed in the majority of cases by oral quinine for 8 weeks, caused rapid disappearance of the parasites with a cure percentage of 18. Twelve intramuscular injections of quinine bihydrochloride in 7-gr. doses every third day, with 30 gr. of quinine orally on the alternate days produced in 94 cases a cure percentage of 27·6, with an average quinine dosage of 1,284 gr. Apart from the above, the sulphate of quinine was given orally, either continuously or intermittently, in four series of cases. In the first continuous series 76 persons received 30 gr. daily for 21 days, followed by 15 gr. daily for 90 days, as advised by Ross & Thomson, with a cure percentage of 52·6, and an average dosage of 1,980 gr. In the second continuous series 190 persons received for 21 days a daily dose of 39 gr., followed by an iron and arsenic tonic, with a cure percentage of 42 and an average quinine dosage of 300 gr. In the two intermittent series the drug was given on two consecutive days weekly for eight weeks as recommended by Stephens. In the first intermittent series 90 men received each 45 gr. daily, in three 15-gr. doses, with a cure percentage of 18·8 and an average dosage of 720 gr. Many cases suffered from vomiting towards the end of this course. In the second intermittent series 113 men received, without toxic symptoms, 30 gr. daily in two 15-gr. doses, with a cure percentage of 30 and an average total dosage of 480 gr. In a total of 663 cases the average cure percentage from a single course of quinine was 32·4. Every benign tertian relapse was, however, followed by an eight-weeks' course of quinine. The percentage of cure following each of four courses of quinine was more or less constant, being 28·9, 20, 28·8 and 20 respectively; or 24·4 on the average. The cure percentage of two eight-weeks' courses, if considered together, is about 45, while that of Ross & Thomson for four months is 52·6, a difference which is considered to be within that of chance distribution. Sixteen such courses would theoretically be necessary for a cure. Regarding the percentage of cure for malignant tertian infection, of 131 persons so diagnosed in the medical history sheets [the relationship of this series to the 102 cases bearing crescents similarly reported being uncertain] 13 relapsed.

The causes determining the partial refractoriness of certain persons to cure by quinine is explained on the same lines as is the general refractoriness of the benign tertian parasite, namely, by the concentration of quinine in the portal system, a purely chance distribution of the parasites determining whether or not they enter the portal area during their vulnerable stage.

One of the most interesting conclusions reached in the paper is the high value attached therapeutically to cinchona febrifuge. Since the use of the total alkaloids of cinchona would relieve the excessive drain occurring in the cinchona plantations in India, the result of the sole use of quinine, and since Prain & Waters have considered it as good as, or better than, quinine weight for weight, it was determined

to test its value under the exceptionally satisfactory conditions regarding possible reinfection which prevail at Dagshai. Two series of cases were observed. In each the daily dose was 21 gr. given continuously, in the first series to 53 men for 21 days and in the second series to 57 men for 10 days. The cure percentage in the first series was 50·9 and in the second 52·6, or exactly the same as occurred in Ross & Thomson's four-months' course. The treatment, which was in tablet form, was popular, without cinchonism or gastric symptoms, and at one-twentieth of the cost of Ross & Thomson's. It is stated, while dealing with quinine, that only in one series did approaching demobilization, with its accompanying possibility of the unauthorized taking of quinine to make cure certain, possibly vitiate conclusions. This point is not mentioned when considering cinchona febrifuge. Clearly their importance, clinical and financial, renders essential the repetition and amplification of these experiments on cinchona febrifuge.

(ii) Apart from the data mentioned in (i) Acton points out that Hodgson has shown that flagellation of the male, with consequent fertilization of the female, gametocyte only occurs while the wet bulb temperature lies between 18° and 22° C., and that prophylactic measures are consequently only required when these conditions are fulfilled. Quinine, if used for this purpose, should be administered in curative doses, that is in doses of 10 or 12 gr. daily, or at most every other day, for seven months. On account of the expense, quinine should be used for troops as a prophylactic measure only when they are on the march or in temporary camps. He advocates that the money thus saved should be spent on electric fans, clearly considering them as sufficing to keep off mosquitoes, as they certainly suffice to convert discomfort into comfort.

[To avoid confusion in the minds of those who refer to the original, it may be pointed out that the reference in (ii) to sections 2, 5 and 6 of (i) as of date 1919 refers to the date of editorial receipt and not to that of publication; that the well-known *Indian Medical Gazette* is camouflaged as the Indian Medical Journal, and the equally well-known Professor Stephens as Stephen; that the portal vein does not carry blood from the intestine to the spleen; that in a vulgar fraction the numerator does not lie below the line; that on p. 857 the figures 2·89, 2·88 and 3·44 should read as 28·9, 28·8 and 24·4 respectively, and that the graph mentioned there is missing, at least from the abstractor's copy. The creation of the new office of Director of Medical Research in India, the holder of which has the editorship of this Journal as part of his duties, has been followed by the bringing of the Journal to date. It can confidently be expected that the energy which has got rid of heavy arrears will presently show itself in more detailed proof reading than the overburdened editor has always found possible in the past.]

C. L.

Ross (Ronald). **Observations on the Principle of Repeated Medication for curing Infections.**—*Brit. Med. Jl.* 1921. July 2. pp. 1–4.

This paper should be read in its entirety. Only a few points can here be selected for notice. There is salutary insistence on the persistent confusion, under the term "immunity," of two entirely different matters, namely, destruction of parasites and compensation of the ill effects which they have caused to their hosts. There is prominence

given to the Single Dose Reduction Rate, which implies that a single dose of medication—in this case quinine—will in nearly all cases reduce the parasites by a definite number or a definite fraction. The dose of quinine recommended on these lines is a 10 gr. daily dose carried out rigorously for 12 weeks—a dose found to suffice in the large majority of cases. In the matter of large doses it is pointed out that the organism subjected to them appears, so to speak, to sense risk of poisoning, and throws them out with increased rapidity, so that the amount of quinine actually circulating is lessened, not increased, by heroic doses. Similarly in the case of the development of resistance to quinine such as the brothers SERGENT have reported [this *Bulletin*, Vol. 18, p. 96] Ross considers that it is probably evidence of an excessively rapid excretion of quinine or its metabolite by the host, rather than of a tolerance to the drug developed by the parasite. [In the SERGENT's case the resistance to quinine was, however, retained in full during the passage of the strain through three canaries in all, and in lessened degree through a fourth.]

C. L.

BRAHMACHARI (U. N.). **The Treatment of Malarial Fever in Individuals Susceptible to Attacks of Blackwater Fever by Intravenous Injection of an Antihaemolytic Quinine Solution.**—*Indian Med. Gaz.* 1921. June. Vol. 56. No. 6. pp. 203–205.

The following observations are based on experiments, the numbers of which, when stated, do not in any case exceed three. The haemolytic properties of quinine and its salts, as tested in vitro, are exhibited in decreasing degree by the bihydrochloride, the hydrochloride, the bisulphate, the sulphate, and the alkaloid. The alkaloid is not absorbed as such when given by the mouth, being converted into one of the chlorides. It is necessary, therefore, to give the alkaloid intravenously. This may be effected by the following solution :—

Quinine alkaloid	5 gr.
Alcohol	50 minims.
Urethane	3 gr.
Calcium chloride	7·5 gr.
Glucose	300 gr.
Normal saline	200 cc. (0·85 % solution of NaCl in distilled water.)

Calcium chloride and glucose help solution of the alkaloid, besides being cardiac stimulants. It is also hoped that they will inhibit any haemolytic action of the quinine, and any haemolysis appearing in blackwater fever. Since 1 cc. of this solution added to a " haemolytic system " produces no haemolysis, it is claimed that the solution is actively antihaemolytic. Further, its injection is said to cause no fall of systolic blood pressure, and it is suggested that it be given a trial in blackwater fever.

C. L.

BASS (C. C.). **The Standard Treatment for Malaria.**—*Public Health Rep.* 1921. July 1. Vol. 36. No. 26. pp. 1502–1504.

In order to popularize Bass's standard treatment of malaria [this *Bulletin*, Vol. 18, p. 97], arrangements have been made to issue quinine sulphate in the form of an eight-weeks' treatment, consisting of 112 capsules of 3, 4 or 5 gr., one or two capsules of the different

values being taken according to age. For children under eight "it is prepared in aromatic syrup of yerba santa, each teaspoonful containing the desired dose according to the doses recommended in the Standard Treatment for different ages."

C. L.

Hylkema (B.). [In Dutch and English.] **De ontwikkeling van de parasiet der quartana in de *Myzomyia ludlowi* en haar overbrenging op den mensch.—The Development of the Parasites of Quartan Malaria in the *Myzomyia ludlowi* and their Transmission on Man.**—*Meded. Burgerlijk. Geneesk. Dienst in Nederl.-Indië.* 1920. Pt. 6. pp. 50–99. With 2 charts and 1 plate.

This is a particularly full and careful account, with excellent references to relevant literature, of experiments showing that in Belawan [N.E. Sumatra] *Anopheles ludlowi* can regularly be infected with the quartan parasite to the extent of 20 per cent., and occasionally to 50 per cent. The insect becomes infective 11 to 13 days after the infecting feed. The quartan cysts are smaller than those of subtertian to begin with, and do not grow so quickly, but the two species of cysts cannot be distinguished *inter se* by their pigment. The susceptibility of *A. ludlowi* for quartan infection, but not for subtertian, decreases with length of captivity (= age), and, as with subtertian, the growth of the cysts is delayed by the same cause.

A. A.

Rodenhuis (Y.). **Dubbel- en superinfectie bij malaria tertiana.** [Double and Super-Infection in Benign Tertian Fever.]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1921. Vol. 61. No. 2. pp. 123–129. With 1 coloured plate.

In cases of malign subtertian, double infection of the red blood corpuscles is a well-known phenomenon; but it is less common in benign tertian fever. The author mentions five cases of the last disease in which red blood corpuscles containing two or three, or even up to six, parasites were found to be present in an unusually high number. All these cases were relapses after a latent period of some duration. Besides a generation of young parasites, the blood films showed older schizonts, segmentation forms and many gametocytes. Of the infected erythrocytes, the author found up to 21 per cent. with double or triple infection. Red blood corpuscles containing gametocytes showed young parasites besides these up to 17 per cent. of their total number.

These percentages are far higher than could be expected on account of the chance calculation the author gives. He therefore thinks that a certain category of red blood corpuscles is more susceptible to malarial infection than the others, and concludes that "in cases where relative immunity does exist (spring relapses in Europe or after latent periods) benign tertian fever can cause many double infections by accumulation of young parasites in a proportion of the red blood corpuscles which has lost immunity. To this proportion belong the already infected ones (containing gametocytes), the granular basophiles, and possibly the polychromatophiles.

"If entering into an erythrocyte containing a gametocyte, the merozoite may happen to get into the protoplasm of a female gametocyte, in this way probably destroying the last. Development of

schizonts beside a gametocyte may give pictures much resembling parthenogenesis" [see this *Bulletin*, Vol. 11, pp. 1–3].

The occurrence of double infection proves to be less pathognomonic of subtertian fever than hitherto it was supposed to be.

[Some details of the plate are not convincing.—C. L.]

W. J. Bais.

i. Levy (Moise D.). **The Wassermann Reaction in Malarial Fevers.**—*Amer. Jl. Trop. Med.* Baltimore. 1921. Sept. Vol. 1. No. 5. pp. 313–317.

ii. St. John (J. H.). **The Wassermann Reaction in Malaria.**—*Ibid.* pp. 319–329.

These writers reach opposite conclusions. Levy, in 50 cases, found four reacting positively, although without history or signs of syphilis, but he believes that the men were syphilitic. St. John found a positive reaction in 4·9 per cent. of 101 cases of acute malaria, but none in 16 chronic cases, all without evidence of syphilis, and considers the former as "due to an undetected increase in the anti-complementary factor."

C. L.

Tempelaar (H. Ch. G.). **Bijdrage tot de morphologie der malaria-parasieten.** [Contribution to the Morphology of the Malarial Parasites.]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1921. Vol. 61. No. 2. pp. 130–137. With 1 coloured plate.

Vital fixation of a blood film is possible by exposing it immediately to the vapour of osmic acid, developing from a half per cent. solution of this acid on the addition of a few drops of concentrated acetic acid. The parasites, after staining in the usual way, show features different from those obtained after application of the usual fixing methods. The author considers the characteristics of the parasite which do not appear after osmium fixation (contracted shape of the parasite, presence of vacuoles) as artificial products caused by the death of the protoplasm. In the osmium films the protoplasm of the parasite appears divided in the erythrocyte. For further details the original should be seen.*

W. J. Bais.

Bertarelli (E.) **Tentativo di transmissione della malaria al macaco.**—*Ann. d'Igiene.* 1920. Nov. Vol. 30. No. 11. pp. 689–690.

Between July and October 1918 the writer took advantage of the possession of a female ape (*Macacus cynomolgus*) to make three attempts to transmit directly the infection of malaria by the injection of citrated blood taken from patients. The first two patients selected were infected with the tertian parasite; the third case was one of aestivo-autumnal fever. In the first case 12 cc. and in the second case 15 cc. of citrated blood were administered intravenously; in the third case the infected blood was introduced first into the vein and afterwards into the peritoneum. Although in all three cases the human blood was seen to contain parasites, asexual as well as sexual forms, none of the experiments was successful in transmitting infection.

J. Rosslyn Earp.

* The osmic acid vapour method of fixation was described by Minchin in 1909 (*Quart. Jl. Mic. Sci.*) and may be of earlier origin.—Ed.

Martini (E.). **Die biologische Malariabekämpfung in Mazedonien.** [Biological Antimalaria Work in Macedonia.]—*Ztschr. angew. Ent.* Berlin. 1921. Feb. Vol. 7. No. 2. pp. 225–286. With 7 text figs. [Summarized in *Rev. Applied Entom.* 1921. June. Vol. 9. Ser. B. Pt. 6. pp. 91–92.]

According to the summary cited this paper describes comprehensively the antimalaria measures undertaken on the German Balkan front in 1918 up to the time of the German retreat. The commonest Anopheles was *A. maculipennis*, the larvae of which were found in all kinds of water where there is no open current, and regularly associated with *Culex hortensis*. Next in abundance was *A. palestinensis*, particularly in the warm region of the lower Varda, usually associated with *Culex mimeticus*; females of *palestinensis* sometimes hibernate. *A. bifurcatus* was not very numerous; it was observed up to 5,000 feet. *A. sinensis* var. *pseudopictus* was captured only once. *A. plumbeus* also occurs. The Culicines observed are mentioned severally and specifically.

A. A.

Stanislas. **Contribution à l'étude de la lutte anti-paludique en Macédoine.**—*Arch. Méd. et Pharm. Milit.* 1921. Apr. Vol. 74. No. 4. pp. 427–429.

On the French military line—Ycnitze-Vardar-Ostrovo—the splenic index was obtained in twenty-five villages, and regular quininization was established in the five of them most severely affected. Antilarval measures were instituted about these same five villages, namely, canalization, regularization of banks of streams, removal of scrub, and petrolage. In some cases the effects in mosquito reduction were rapid and striking, but, so long as irrigation is carried out with its present disregard of care, complete removal of breeding places is impossible. Tents were screened, mosquito nets provided, and quinine administered, the last checked by surprise tests of the urine, which showed that the percentage of those taking quinine properly rose from 72 at the beginning of the operations to 95 at their end.

As a result, the members of the malaria commission remained without a single case of malaria. No reference is, however, made to the health of the French troops in general.

C. L.

Génévrier (J.). **Prophylaxie du paludisme en milieu militaire.—De la stérilisation du paludisme par les injections intraveineuses de quinine.**—*Bull. Soc. Méd. Milit. Fr.* 1921. Apr. 21. Vol. 15. pp. 137–145, avec remarques de Visbecq, Pouy, Wissemans. [Summarized in *Bull. Inst. Pasteur.* 1921. July 30. Vol. 19. No. 14. p. 524.]

Génévrier claims that the best way of preventing malaria is by sterilization of carriers, and that this is most satisfactorily effected by intravenous injection of quinine, which is effective in the first stage (lasting for the first 10 or 12 days after infection), useful in the second stage, and ineffectual in the third stage, that is, when visceral lesions have become established. Edm. Sergent, in abstracting the paper, objects to the division into stages of an infection which is throughout a septicaemia. Visbecq notes that to abandon oral medication is impossible, and Pouy insists that the oral and intravenous routes have their respective places.

C. L.

YOFÉ (Hillel). **Proposals for Antimalaria Work in Palestine.**—*Internat. Jl. Public Health.* 1921. Sept.–Oct. Vol. 2. No. 5. pp. 478–487.

This is a piece of reasoned and reasonable special pleading by one whose thirty years' work in Palestine has left him with the certainty that his country has a great future if malaria can be eliminated. He formulates a scheme of control, of which the essential elements are (1) a trigonometrical, malarial and geological survey aimed at correlating the requirements of sanitation and agriculture; (2) the collection of morbidity and mortality statistics; and (3) an Anopheline survey in all its phases. A sanitary engineering and a sanitary medical service, adequately equipped and working with agricultural authorities, could utilize the favourable natural drainage of the country. By taking advantage of the Turkish law that unused marsh land becomes the property of him who drains it, there would be strong inducement for the finding of the funds necessary for this purpose. Those who own the land should pay for the free distribution of quinine by the State, the drug having in the writer's experience, proved to be an effective prophylactic. For the rest of the requisite funds an appeal is made to those who have at heart the welfare of Palestine and of the human race generally.

C. L.

i. LÓPEZ (J. A.). [Malaria and Tuberculosis.]—*Semana Médica.* Buenos Aires. 1921. May 12. Vol. 28. No. 19. p. 550. [Summarized in *Jl. Amer. Med. Assoc.* 1921. July 30. Vol. 77. No. 5. p. 412.]

ii. SUGIYAMA (S.). On Nervous Otalgia preceding the Paroxysm of Malaria.—*Taiwan Igakkai Zasshi* (*Jl. of the Med. Assoc. of Formosa*). 1921. May 31. No. 216. (English summary, p. 2.) [In Japanese.]

iii. JIMÉNEZ (R.) & PITTALUGA (G.). [Malaria recurs after Splenectomy.]—*Arch. de Cardiología y Hematología.* Madrid. 1921. June. Vol. 2. No. 6. p. 233. [Summarized in *Jl. Amer. Med. Assoc.* 1921. Sept. 10. Vol. 77. No. 11. p. 896.]

i. "López relates that in his experience with three generations of army recruits, he has frequently found that young men with a history of old malaria did not respond properly to quinin, and certain minor signs suggested superposed tuberculosis. Applying Vitón's tuberculin therapy with the minutest doses, supplemented with an antituberculosis vaccine, these men recovered, throwing off both the symptoms suspicious of tuberculosis and the malaria as well."

ii. "The author . . . . describes in detail two cases of his latest experience who had severe ear-ache as a prodrome to malaria, that disappeared together with the paroxysm upon chinin treatment."

iii. "The woman of 31 had been apparently long cured of the tertian malaria but it flared up anew after removal of the enlarged spleen."

C. L.

PLAGUE.

Low (R. Bruce). **Reports on Public Health and Medical Subjects. No. 3. The Progress and Diffusion of Plague, Cholera and Yellow Fever throughout the World, 1914-1917.** With an Introduction by Sir George Newman.—276 pp. 1920. Ministry of Health. London: Published by His Majesty's Stationery Office. [Price 7*s.* 6*d.* net.]

The section referring to cholera has been reviewed in a special article [this *Bulletin*, Vol. 18, pp. 69–77]. Yellow fever does not come within the scope of this section.

The population of India, according to the 1911 census, was 315,156,396, and from that year to the end of 1917 no less than 2,970,318 persons died of plague in India. The disease first appeared in Bombay in the autumn of 1896, introduced probably from Hong Kong, where there was an epidemic. From the date of the first infection to the end of 1917 the reported deaths amount to 9,841,396, but many deaths in outlying villages were most probably never reported. The village watchman, whose duty it is to report deaths, would, quite apart from his lack of medical knowledge, be anxious to conceal from sanitary officials the fact that plague existed, and, with his fellows, would make investigation difficult. The greatest intensity was reached in 1907. In that year 1,315,892 persons died, a rate per mille of 5·16. Of the total deaths 74·4 per cent. occurred in three presidencies:—(1) the Punjab; (2) Bombay; and (3) the United Provinces. The combined population of these three areas is about 98 millions. The distribution of population and the death-rate were:—

	Population 1911.	Plague Deaths 1896–1917.
The Punjab	24,187,750	2,862,852
Bombay Presidency	27,084,317	2,257,682
United Provinces	47,182,044	2,201,811
	98,454,111	7,322,345

Much has been done by the Plague Commissions, the Royal Society, and the Lister Institute, to investigate the various features of the disease and to provide the individual members of the Indian Medical Service with powers to apply their knowledge in the treatment, eradication and prevention of the dread malady caused by *B. pestis* (Yersin). Why, then, does plague show "no obvious tendency, up to the time of writing, to cease its ravages in India?"

There is no doubt that the people themselves are chiefly responsible for lack of success in coping with the infection, which is endemic in certain areas in Northern India. Sanitary regulations are regarded, especially by Hindus and Mohammedans, whose women are kept behind the purdah, as affronts to religion, caste or custom. Political agitators make use of these prejudices, due in the main, to ignorance, to raise up opposition, often using methods which they must know to be wrong and without truth. Destruction of rats, so necessary to the stamping out of plague, is opposed, although they are killed to protect property by low-caste men. The Jain, the Buddhist and most Hindus, while personally refraining as part of their religion from taking the life even of an insect such as the plague flea, have no rooted

objection to killing when it is done by a Dôm or pariah. Isolation of the sick and removal to hospital is resented, and even medical women have been mobbed. "No doubt the education of the native people is progressing, but it has not gone far enough as yet to effect much improvement in the incidence of fatal plague in India, judging by the mortality returns."

Dr. R. St. John Brooks has investigated the effect of hot weather on epidemic plague. His conclusions are: "That in India plague does not maintain itself in epidemic form when the temperature rises above 80° F. accompanied by a saturation deficiency of over 0·30 of an inch." By "saturation deficiency" is meant: "the difference between the actual tension of aqueous vapour present in the atmosphere at the temperature in question and the tension of aqueous vapour that would be present in a saturated atmosphere at the same temperature." There is, however, nothing to prevent plague spreading in India or elsewhere when once rats are infected and fleas have carried that infection to man in unsanitary ports and other areas. Moreover, septicaemic and pneumonic plague, the most dangerous forms, are independent, once started, of rats or fleas. The statistics of plague and the value of anti-plague vaccines have been fully noticed in this *Bulletin*, Vols. 14, 15 and 16. In anti-plague vaccines we have a medical weapon which can and should be used, not only for prophylaxis, but for treatment.

J. H. Tull Walsh.

Flu (P. C.). **Enkele epidemiologische waarnemingen over pest.** [Some Epidemiological Observations on Plague.]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1921. Vol. 61. No. 3. pp. 263–287.

Epidemiological observations were made by the author during outbreaks of rat plague at Palembang, Tandjong Priok, Batavia and Weltevreden in 1920.

He did not succeed in demonstrating the infection either in living rats caught round the ports or in the fleas they were carrying, notwithstanding rat plague was present, but only in corpses of rats found in the godowns. Cutaneous inoculation was found to be the most practical way to show the plague infection in material from rat corpses or from fleas.

From the epidemiological facts at Palembang the importance of rice transport for the spreading of rat plague is apparent. Till now the rats themselves have been considered to be responsible for the spread of the disease, but the author never found any dead rats between or in the rice bags in many godowns inspected. The corpses were always found under the heaps of bags between the floor of the godown and the plank floor on which the rice bags are piled up.

The author thereby was led to the presumption that infected fleas might play a more important part in plague spread by rice transport than infected rats. In many godowns guinea-pigs were used as flea traps to gather any fleas that might be living in or on the goods stocked. Rat fleas (*Xenopsylla cheopis*) were caught in every case, more in godowns with goods attractive to rats (rice, flour, dried fish) than in those with iron work, etc. Of 701 fleas caught in this way in different godowns after the outbreak of rat plague at Tandjong Priok 69 were found to harbour the plague bacillus. The fleas crawl over

the bags of rice and hide between the meshes, or may bore their way into the bags. They can easily be transported in rice, as infected fleas may go 10–14 days without food (Hoesen, Swellengrebel). This confirms the author's views, but does not make the prospects of plague combating more hopeful, as fleas are far more difficult to control than rats.

The author therefore made another series of experiments to find out whether our most powerful means in plague quarantine at present, namely claytonage, kills the fleas with sufficient certainty. In bottles the fleas die immediately in an atmosphere containing 4 per cent. sulphurous acid, within one hour in half per cent. of the same. If a rice bag in the bottle offered a hiding place to the fleas many of them were found alive after two hours. In practice similar circumstances exist, and experiments made during the claytonage of a vessel had much the same results. The rice cargo also seems to absorb an important part of the gas introduced into the ship.

The author thinks that improvement in plague quarantine is necessary: (1) by using more powerful poisons in the place of sulphurous acid in claytonage, *e.g.*, prussic acid, which, however, is far more dangerous to man, and only practicable if the necessary precautions are taken; (2) by rat-proofing godowns and other buildings in the vicinity of the ports. The godowns should also be built in a way that makes it possible to close off the whole building without great expense, so as to make treatment with gaseous insecticides practicable.

W. J. Bais.

Dold (Hermann). **Die periodischen Schwankungen der Rattenpest in Schanghai, ihre Beziehungen zur Temperatur und Feuchtigkeit.** [The Periodic Variation of Rat Plague in Shanghai in relation to Temperature and Humidity.]—*Ztschr. f. Hyg. u. Infektionskr.* 1921. Mar. 5. Vol. 92. No. 1. pp. 1–8. With 7 charts in text.

The author's work is given in useful "tables" and charts, which present clearly the relations set out for investigation. It will suffice to reproduce Table I and the charts for 1909 and 1914, since, in each chart, there is, in the months April to November, a marked fall in rat plague, with a rise in temperature and moisture.

Table I.

Year.	Number of rats found dead and examined.	Number of Infected Rats.	Per cent.	Cases of Human Plague.
1909 ..	17,364	187	1·08	0
1910 ..	19,559	249	1·27	6
1911 ..	14,929	138	0·90	0
1912 ..	14,988	95	0·64	18
1913 ..	13,333	122	0·90	2
1914 ..	12,782	186	1·44	23

To these may be added one chart compounded by Dr. Dold from the Report of the Indian Plague Commission (for *M. rattus* and *M. decumanus*).

J. H. T. W.

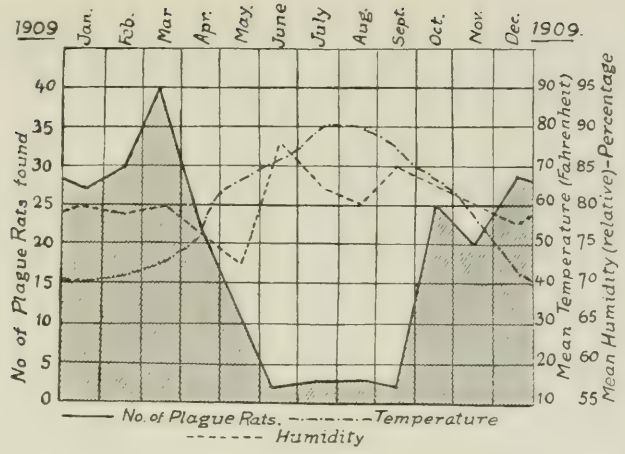

1909
Jan. Feb. Mar. Apr. May. June July Aug. Sept. Oct. Nov. Dec.
1909.
No of Plague Rats found
Mean Temperature (Fahrenheit)
Mean Humidity (relative)-Percentage
No. of Plague Rats. -.-.-.-Temperature
------- Humidity

Chart 1.—1909.

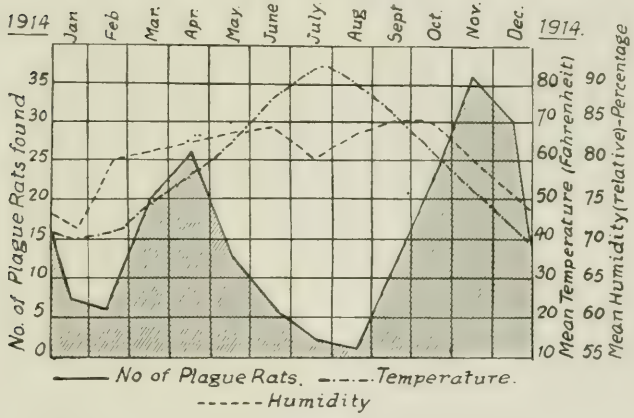


Chart 6.—1914.

Charts 1 and 6.—Showing monthly findings of Plague-infected Rats in Shanghai during 1909 and 1914, compared with Mean Monthly Temperature and Humidity. [Dold.]

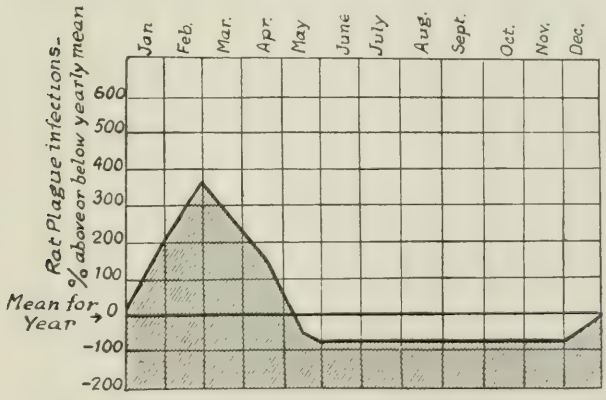


Chart 7.—Showing monthly findings of Plague-infected Rats (*M. rattus* and *M. decumanus*) in Bombay, expressed as a percentage above and below the mean for the year. [Based on curves of the Indian Plague Commission.]

Cragg (F. W.). **The Distribution of the Indian Species of the Genus *Xenopsylla*, with reference to the Immunity of Certain Areas from Plague Epidemics.**—*Indian Jl. Med. Res.* (Special Indian Science Congress Number, 1920.) pp. 29–34.

Altogether 4,270 fleas were examined by Major Cragg. They came from 18 areas in India. Of the total number 64·5 per cent. were *cheopis*; 29·8 per cent. *astia*; 2·7 per cent. *brasiliensis*; and 2·89 per cent. belonged to the genus *Ceratophyllus*. These last were examined by Mr. Rothschild, who thinks they are probably not *fasciatus*. In the Punjab, *cheopis* predominates:—Rawalpindi, 92·8 per cent.; Ludhiana, 93 per cent.; Sialkot, 85 per cent.; Jullunder, 73 per cent.; and Jhelum, 86 per cent. From each of these stations a few *astia* were sent [do not bite man; see this *Bulletin*, Bacot, Vol. 16, p. 45]. *Brasiliensis*, which also does not readily bite man, was absent. All the fleas received from Multan belonged to *astia*. Of the doubtful species of genus *Ceratophyllus* [? *ainsus*, N. and E. Asia; does not bite man (Bacot)], 14 per cent. were present in the Jullunder batch of 116 fleas. Out of 784 fleas from Bombay city, 49·8 per cent. were *astia*, 49·5 per cent. *cheopis*, the rest *brasiliensis*. In Poona, a colder climate than Bombay, 87 per cent. out of 466 fleas were *cheopis*, the remainder *brasiliensis*. The figures for Karachi are: 85 per cent. *cheopis*; the rest *astia*. Those of a small batch of fleas from Akyab, Burma, which has never suffered from plague, 98 per cent. *astia*.

[The only flea mentioned by Major Cragg not found in Bacot's list is *X. vigetus*, Rothschild, 1909 (*brasiliensis*, Jordan and Rothschild, 1908).]

For distribution of species for the U.S.A., see this *Bulletin*, Vol. 17, p. 389 (McCoy).

J. H. T. W.

Teissier (P.). **A propos d'un cas de peste bubonique.**—*Bull et Mém. Soc. Méd. Hôpit. de Paris.* 1921. Feb. 10. Vol. 45. No. 4. pp. 98–101.

——, Tanon (L.), Gastinel (P.) & Reilly (I.). **Valeur diagnostique de l'hémoculture dans la peste bubonique; fréquence de la bacillémie pesteuse.**—*Ibid.* Feb. 17. No. 5. pp. 136–138.

— –, —— & ——. **Remarques sur l'épidémie de peste observée è l'hôpital Claude Bernard.**—*Ibid.* pp. 138–144.

Guinon & de Pfeffel. **Quelques aspects cliniques de la peste chez l'enfant.**—*Ibid.* Feb. 25 No. 6. pp. 215–220.

These papers have reference to the epidemic described in this *Bulletin*, Vol. 17, p. 392; the cases are of the same kind.

i. This was a case treated by M. Teissier, which, in spite of the fact that no bacilli were found by blood-culture, he believes to have been plague. The clinical symptoms were those of plague, with buboes in the right groin. On the skin of the abdomen were numerous marks of flea-bites.

ii. The writers found that even in mild cases of plague an early bacillaemia is nearly always present, but that where it is present in the

later stages of the disease prognosis is bad. They give the following table as a résumé of their work :—

Names.	Blood Culture.	Temperature.	Day of illness.	Course of disease.
Des	+	40°	3rd day	Septicaemia. Death.
Ausch	+	40°	2nd day	Severe. Delirium. Cure.
Mart (woman) ..	—	40°	2nd day	Mild. Blood-culture not preserved after 48 hours.
M.	+	39°	4th day	Moderate.
C. (daughter) ..	—	39°	4th day	Mild.
C. (mother) ..	+	40°	4th day	Mild.
Ver.	+	38·5°	3rd day	Mild.
Qu.	+	41°	4th day	Septicaemia. Pulmonary and hepatic complications. Death.
B.	—	38°	15th day	Mild.
Pail	+	40°	9th day	Severe. Cure.
Del.	+	37·5°	12th day	Mild.
Pic.	—	39°	11th day	Bubo with extensive suppuration (associated with streptococci and staphylococci). Cure.

iii. The authors are mainly concerned with prophylaxis, to which was due the limited extension of the epidemic. They point out that : "The epidemic of plague which broke out in Paris a few months previously had, owing to prophylactic measures and especially to vaccinations, but a limited extension."

iv. Practically presents the same features as described in the cases referred to on page 393 of Vol. 17 of this *Bulletin*.

J. H. T. W.

Cintra (A. P. de Ulhôa). **Sobre a ultima epidemia de peste em S. Paulo.** —*Bol. Soc. Med. e Cirurg. de S. Paulo.* Brazil. 1920 & 1921. Oct.–Feb. Vol. 3. (2nd Ser.) Nos. 8–12. pp. 358–361.

Records particulars of a somewhat anomalous epidemic at first thought to be influenza pneumonica. The virulence of the infection, with rapid mortality, pointed to pneumonic plague. Examination of several fatal cases resulted in the finding of a Gram-negative germ resembling the cocco-bacillus of plague (Yersin). Experiments on guinea-pigs produced death in 3–6 days and, except for agglutination, the bacilli were like those of plague [possibly modified in pneumonic plague and more virulent than strains from bubonic plague]. About the same time Motta (I.) also described "plague-like bacilli," and Smillie (W. G.) found in rats dying in S. Paulo a variety of plague-bacillus. [See this *Bulletin*, Vol. 17, p. 398.]

J. H. T. W.

Boyd (Mark F.) & Kemmerer (T. W.). **Experience with Bubonic Plague (Human and Rodent) in Galveston, 1920.**—*Public Health Rep.* 1921. July 29. Vol. 36. No. 30. pp. 1754–1764. With 2 text figs.

These outbreaks in Galveston and other gulf ports have already been noticed [see this *Bulletin*, Vol. 17, pp. 387–9 (McCoy) ; pp. 389 & 394 (Levy & McMicken ; Levy)].

This report contains nothing of further importance.

J. H. T. W.

Araujo (Eduardo). **Determinações cutaneas da peste. Estatistica. Aspecto clinico. Pathogenia.** [Clinical and Pathological Aspects of Cutaneous Symptoms of Plague.]—*Brazil Medico.* 1921. July 16. Year 35. Vol. 2. No. 1. pp. 1–4.

Among 827 cases of plague admitted to the Monte Serrat hospital, 16 presented skin troubles of various kinds: pustular eruptions, phlyctenulae and bullae, etc. Yersin's bacillus was isolated [see this *Bulletin*, Patane, Vol. 13, p. 326, and Alvarado & Baca, Vol. 16, p. 46].

J. H. T. W.

Schut (H.). **Over eenige gevallen van longenpest.** [Some Cases of Pulmonary Plague.]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1921. Vol. 61. No. 1. pp. 65–70. With 4 charts.

Reports on five cases, all ending fatally. The author noted temporary improvement after administration of neosalvarsan (0·45 gm. on two consecutive days) and digalen (intramuscular injections of 1 cc. three times per day). He recommends the trial of this medication, combined with infusion of glucose solution into the veins to guard against possible heart failure.

W. J. Bais.

Harrison (W. T.). **Report of a Human Plague Case in San Benito County, Calif.**—*Public Health Rep.* 1921. Sept. 9. Vol. 36. No. 36. pp. 2219–2220.

Though this is an ordinary plague case it presents points of interest and lessons for all who work in infected regions. The source of infection was an old squirrel plague focus in the Bitterwater Valley, San Benito County [see this *Bulletin*, Vol. 17, p. 387 (McCoy) and Harrison, Vol. 16, p. 433].

An early diagnosis was made by Dr. O'Bannon, of Hollister, and large doses of anti-plague serum, obtained from F. J. Lackenbach, of San Francisco, were injected 68 hours after the onset. Owing to this prompt treatment the patient, a boy eight years old, recovered. This is the more satisfactory as the bacilli settled in the glands of the axilla and scapular region [see McCoy, *l.c.*; also Monziol and Colignon, Vol. 16, p. 43].

Serous fluid from the oedematous area produced no bacilli; but when the swelling of the shoulder subsided Yersin's bacillus was cultivated from juice drawn from the enlarged lymphatic glands. Guinea-pigs inoculated intraperitoneally died within 48 hours.

J. H. T. W.

Montfort. **Un cas de peste authochtone en Nouvelle-Calédonie.**—*Ann. de Méd. et de Pharm. Colon.* Paris. 1921. Jan.–Feb.–Mar. Vol. 19. No. 1. pp. 72–75.

A case of bubonic plague, given with full details. Yersin's bacillus was found, and mice died with signs of murine plague, after inoculation with fluid from the inguinal bubo.

J. H. T. W.

Kunhardt (J. C. G.) & Chitre (G. D.). **An Experiment in the Eradication of Plague Infection carried out in the Poona and Adjacent Districts. First Report. (For the Period 1914-1916.)**— *Indian Jl. Med. Res.* 1921. Jan. Vol. 8. No. 3. pp. 409–445. With 13 charts and 5 maps.

—— & ——. **Further Experiments in Plague Prevention carried out at Poona. Second Report. (For the period 1916–1918).**— *Ibid.* pp. 446–489.

These two long reports with useful charts and maps contain confirmation of much of the work presented by various writers to the Special Indian Science Congress in 1919 [see this *Bulletin*, Vol. 16, pp. 49–52].

J. H. T. W.

Uriarte (Leopoldo). **Profilaxis de la peste de Orient. Las ratas y los depósitos de mercaderiás.**— *An. del Depart. Nac. Hig.* Buenos Aires. 1920. Sept.–Oct. Vol. 26. No. 5. pp. 293–318. With 4 text figs. and 20 illustrations.

This is a long and interesting article showing how grain may be stored and protected from rats, either out in the open, while waiting to be transferred by rail, or in warehouses. Similar methods of prevention and rat destruction have previously been noticed in this *Bulletin*, Vol. 16, pp. 49–52.

J. H. T. W.

Kellog (W. H.). **Epidemic of Pneumonic Plague.**— *Amer. Jl. Public Health.* Boston. 1920. July. Vol. 10. No. 7. p. 599. [Cited in the *Jl. Amer. Med. Assoc.* 1920. Aug. Vol. 75. No. 8. p. 563.]

Since this article was written the history of plague in the United States of America has been recorded by Dr. G. W. McCoy, and the epidemic of pneumonic plague is mentioned by him. [See this *Bulletin*, Vol. 17, p. 387.]

J. H. T. W.

Boinet (Édouard). **La peste de Marseille, en 1720 et 1721.**— *Bull. Acad. Méd.* 1921. July 5. Vol. 86. No. 27. pp. 10–15.

Historical, and gives dates of epidemics from 1383 to 1721 ; and gives a quotation from Petronius pointing to a much earlier infection.

J. H. T. W.

CHOLERA.

Crendiropoulo (M.). **Assainissement général, prophylaxie. Rapport concernant des expériences sur les porteurs de vibrions cholériques.** —*Bull. Office Internat. d'Hyg. Publique.* 1921. Mar. Vol. 13. No. 3. pp. 247–255.

The author is Director of the Bacteriological Laboratory, Alexandria, and his work is of importance, especially if compared with Professor G. Sanarelli's experiments with guinea-pigs [see this *Bulletin*, Vol. 16, p. 438]. Rabbits were the subjects of Dr. Crendiropoulo's inoculations.

During the October session of the permanent Committee of the Office of International Hygiene a desire was expressed for further contributions on the cholera carrier. The period of retention of the cholera vibrio in the alimentary canal is of importance, not only as a guide to a rational and efficacious prophylaxis of the disease, but also for reasons of general value. The first step was to find out the best route for introduction of the infection and the amount necessary to render the animal a carrier. The technique was as follows :— After a certain dose of vibrios had been injected by the selected route, and after a given lapse of time, which varied from a few minutes to several months, the animal was pithed. The body was opened and, by means of cotton-wool tampons, separate specimens for culture were taken from the stomach, the duodenum, the jejunum, the ileum, the appendix, the caecum and the large gut. At the same time the bile, blood, urine, and often the liver, spleen, kidneys, suprarenal capsules and the lungs were found to be infected.

At first the author used vibrio No. 136 " El Tor ." from a patient of 1913, but later preferred a more virulent strain from the stools of an Annamite patient, brought to Alexandria by the " Messageries Maritimes," in 1916.

Hypodermic route.—By this route, speaking generally, the vibrio does not penetrate the body ; even in lethal doses the author only found vibrios in the appendix in one case out of 12. The animals died of intoxication, not of septicaemia.

Intraperitoneal route.—The vibrio killed the rabbits with certainty in a dose of 1/5 of the culture ; but with a much smaller dose it was found in the alimentary canal. Injecting 1/15, one was sure to find it in the intestines, and often in the bile and the blood. Thus out of four rabbits inoculated with this dose, and killed at the end of four hours, he found the vibrio four times in the duodenum, ileum and appendix ; three times in the blood, the bile, the caecum and the large intestine ; and once only in the urine. In four others killed at the end of eight hours there were similar results. When the animals were killed at the end of 48 hours the chance of finding vibrios in such places was less ; and at the end of three days no vibrios were found, although the author repeated his experiments three times. With smaller doses the results were uncertain, but sometimes the vibrio was isolated from the small gut, less often from the bile, and very rarely from the blood and other tissues, even during the first 24 hours. After that period results were negative.

Intravenous route.—Fifty-three rabbits were thus infected. The lethal dose was 1/30 of the culture ; with 1/50 the greater number

survived ; 1/10 killed a rabbit in from 8 to 24 hours. Twelve rabbits receiving this dose gave positive culture results from all organs even when killed as early as 10 minutes after the injection. With 1/50 the positive results were less marked ; but six killed from 15 minutes to 24 hours later showed vibrios in the small intestines, bile, urine, spleen and liver. Once only were all cultures negative. Out of 10 rabbits that received only 1/100, all but one had eliminated the vibrios at the end of one hour.

By the stomach.—Vibrios introduced under normal conditions are killed and rarely pass into the intestine ; ordinarily the animals were left for 12 to 68 hours fasting before the vibrios were introduced by means of a tube. An entire culture made into emulsion with 10 cc. of physiological saline was given. The animals were killed in 30 minutes to two hours. Out of nine experiments seven were absolutely negative ; in two rabbits vibrios were present at the end of one hour ; one retained them alive in the stomach, the other in the appendix, cultures from other organs giving negative results. The fact remains that with rabbits the stomach forms an "almost insurmountable barrier to invasion of the body by *B. cholerae*."

The rectal route.—Cholera vibrios, even in small doses—1/50 to 1/100—pass into the various organs in 75 per cent. At the end of one hour no vibrios were found in 25 per cent.

J. H. Tull Walsh.

Douglas (S. R.). **The Question of Serological Races of *V. cholerae* and the Relation of some Other Vibrios to this Species.**—*British Jl. Experim. Path.* 1921. Apr. Vol. 2. No. 2. pp. 49–57.

Major Douglas, I.M.S., has collected and tested the many varieties of vibrios which cause cholera. Most of them are geographical varieties, differing from type *V. cholerae* in agglutination reaction. Some cause haemolysis in blood culture media. The question of variety of strains has been examined in various records of epidemics noticed in this *Bulletin*, and in the Sectional Editor's "Critical Summary" (Vol. 18, p. 69).

The author's conclusions may be summarized as follows :—

(1) All strains of *V. cholerae* investigated belonged to one serological race, and, as these strains came from widely different sources, it is probable that there is only one serological race of *V. cholerae* [with varieties].

(2) The advantage of using emulsions made by formolizing broth cultures, as recommended by Dreyer, when comparing the agglutinating properties of a series of strains of the same organism, is sharply brought out.

(3) After repeated subcultures, *V. paracholerae* A acquired the property of agglutinating with type serum. This property was not lost when virulence was restored by passage through guinea-pigs.

(4) Of "El Tor" vibrios, A 4 and 5 behaved as type *V. cholerae*. Strain 10 was like *V. paracholerae* A., *V. nasik*, *V. paracholerae* B, and the "El Tor" 35 differed from the type vibrio and from *V. paracholerae* A.

J. H. T. W.

Naamé. **L'adrénaline dans le choléra.**—*Gaz. des Hôpit.* 1921. May 31. Vol. 94. No. 43. pp. 678–679.

The interesting part of this paper lies in the possible explanation of the uselessness of anticholera vaccine as a therapeutic agent. The author thinks that while the vaccine, used as a prophylactic agent,

acts on an individual with healthy suprarenal bodies, sending a normal supply of adrenalin into the circulation, such supply is non-effective, if it ever reaches the blood, during an attack of cholera. The cholera patient tolerates large doses of adrenalin. M. A. MARIE has shown that, in guinea-pigs, adrenalin will neutralize a lethal dose of cholera vibrios, and SAJOUS writes that the endocrine secretions in the blood favour the production of germicide substances and antitoxins. They also encourage phagocytosis (*Presse Méd.*, 1912, Jan. 27). VIOLLE (Le Choléra, pp. 170 and 425) writes :—

"In 1911 Naamé began a new treatment for cholera; he treated his patients with adrenalin, and obtained great success. Doctors in India, and in Italy especially, applied this method and were equally satisfied . . . . . With large intravenous injections of saline, to which adrenalin is added, the algid stage of cholera is, at the present day, best controlled."

[For the effect of cholera toxin on the suprarenal secretion of rabbits, see this *Bulletin*, Vol. 6, p. 41. DÉMÉTRESCU.]

J. H. T. W.

MACKIE (F. P.) & GUPTA (J. C.). **Statistics of the Treatment of Cholera.** —*Indian Med. Gaz.* 1921. June. Vol. 56. No. 6. pp. 201–203.

The professor of pathology and the house-surgeon of the Medical College Hospital, Calcutta, following the treatment inaugurated by Sir Leonard ROGERS, give an analysis of cholera statistics for June to December, 1920, as set out in Table II :—

TABLE II.

Months.	Type of Disease.	Per cent. of Deaths.	Deaths.	Recoveries.	Totals.
June	Mild	..	Nil	10	10
	Moderate	..	3	35	38
	Severe	..	27	6	33
	Totals	37	30	51	81
July	Mild	..	Nil	16	16
	Moderate	..	4	39	43
	Severe	..	10	13	23
	Totals	17	14	68	82
August	Mild	..	Nil	[1]	1
	Moderate	..	Nil	[9]	9
	Severe	..	1	[3]	4
	Totals	7	1	13	14
September	Mild	..	Nil	2	2
	Moderate	..	1	4	5
	Severe	..	3	7	10
	Totals	23	4	13	17

TABLE II.—*continued.*

Months.	Type of Disease.	Per cent. of Deaths.	Deaths.	Recoveries.	Totals.
October	Mild	..	Nil	5	5
	Moderate ..	..	Nil	7	7
	Severe	..	3	4	7
	Totals ..	16	3	16	19
November	Mild	..	Nil	7	7
	Moderate ..	..	2	25	27
	Severe	..	10	7	17
	Totals ..	23	12	39	51
December	Mild	..	Nil	3	3
	Moderate ..	..	Nil	21	21
	Severe	..	Nil	10	10
	Totals ..	..	Nil	34	34
		21	64	234	298

The causes of death were :—

Suppression of urine	25
Cardiac failure	29
Abortion	2
Collapse	10
	64

Tables V and VI also give useful information concerning the specific gravity of the blood and the amount of saline injected :—

TABLE V.

Sp. Gravity.	Totals.	Deaths.	Recoveries.	Sp. Gravity.	Totals.	Deaths.	Recoveries.
Over 1070..	7	1	6	1062..	28	4	24
1070..	5	2	3	1061..	23	5	18
1069..	15	3	12	1060..	11	2	9
1068..	21	10	11	1059..	3	0	3
1067..	9	1	8	1058..	20	1	19
1066..	21	4	17	1057..	8	0	8
1065..	9	1	8	1056..	15	2	13
1064..	15	3	12	1055..	2	0	2
1063..	30	6	24	Under 1055..	17	1	16

TABLE VI.

No. of Pints.	Cases.		Deaths.	Recoveries.
	Intravenous.	Subcutaneous.		
Over 10 pints.. ..	43	Nil	21	22
9 ,, ...	12	Nil	3	9
8 ,, ...	13	Nil	Nil	13
7 ,, ...	6	Nil	1	5
6 ,, ...	23	1	2	21
5 ,, ...	22	Nil	4	18
4 ,, ...	31	1	4	27
3 ,, ...	32	6	5	27
2 ,, ...	33	7	2	31
1 ,, ...	24	27	Nil	24
Nil	Nil	Nil	Nil	32

J. H. T. W.

WALKER (Ronald R.). **The Action and Uses of Kaolin in the Treatment of Asiatic Cholera.**—*Lancet.* 1921. Aug. 6. pp. 273–276.

Refers to work done by Louis H. BRAAFLADT [see this *Bulletin*, Vol. 16, p. 434]. Dr. Walker used kaolin during an epidemic in Foochow (1919), obtaining the material from " the famous Blanc de Chine beds " in the Hing Hwa district.

" A large supply of half-and-half suspension was placed near the patient, and the nurse was told to encourage the patient to take as much as possible . . . . . In all cases food was withheld for 18 to 24 hours, then rice water was allowed, and later milk and rice water. In all cases rectal lavage was done with kaolin solution thickened until it would comfortably pass through the rectal tube . . . . . If the condition of the patient was precarious on arrival at hospital, it was found that subcutaneous bilateral infusion usually restored the patient sufficiently to take kaolin by the mouth. In desperate cases hypertonic salt solution was given intravenously (ROGERS), but no such large quantities as we used before the introduction of the kaolin treatment . . . "

The author carried out some experiments in which rabbits were fed with kaolin, which after 18 hours was found adhering to the walls of the stomach and intestines.

J. H. T. W.

FLU (P. C.). **Onderzoekingen over den levensduur van cholera-vibrionen en typhusbakteriën in septic tanks te Batavia.** [Researches on the Duration of Life of *Vibrio cholerae* and Typhoid Bacteria in Septic Tanks in Batavia.]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1921. Vol. 61. No. 3. pp. 288–293.

The cholera vibrios died sooner in the septic tanks than did the typhoid bacilli inoculated into them. In one series of experiments (Kampong Pintoe Besie) vibrios had disappeared at the end of 24 hours, while typhoid bacilli were present up to the end of the fifth day; in another (Kampong Kebon Djeroek) the vibrios were still living at the end of 24 hours, the typhoid bacilli at the end of 72 hours.

J. H. T. W.

Flu (P. C.). **Onderzoekingen over den levensduur van choleravibrionen en typhusbacteriën in zeewater.** [Researches on the Duration of Life of *Vibrio cholerae* and Typhoid Bacteria in Sea Water.]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1921. Vol. 61. No. 3. pp. 307–316.

The different opinions expressed by authors on this subject are ascribed by the author to differences in climate and sort of water, differences in quantities and strains of cholera vibrios used, and in methods employed in detecting the germs again after the artificial pollution of the sea water. The author made his experiments at Tandjong Priok, the port of Batavia. For the technic used by him the original should be seen. He could not demonstrate any preserving action of sea water on the cholera vibrio; this, and the *Bacillus typhosus* as well, disappeared from infected sea water within four days. Yet this period necessitates precaution against sea water that may be freshly contaminated, such as water of ports in times of epidemics.

W. J. Bais.

Fukuhara (Y.). **Die weitere Anwendung unserer neuen Methode zur Wertbestimmung der antiinfektiösen Cholera- sowie Paratyphus-sera.** [Further Application of a New Method of Estimating the Value of Anti-Cholera and Anti-Paratyphoid Sera.]—*Cent. f. Bakt.* 1. Abt. Orig. 1921. July 8. Vol. 86. No. 6. pp. 450–458.

The cholera serum is compared with a standard serum, the minimum lethal dose of which is known for the animal under experiment. The serum to be tested is mixed with the standard serum in various proportions and its effect on the animal observed. If, for example, the tested serum in such a mixture producing the death of the animal in 24 hours is 0·001 cc., then that serum contains 1,000 immunity units in 1 cc.

J. H. T. W.

Snapper (J.). **Die Zersetzung von Blut und Blutfarbstoff durch Cholera- und Tor-Vibrionen.** [Decomposition of Blood and Haemoglobin by Cholera and "El Tor" Vibrios in Blood Cultures.]—*Cent. f. Bakt.* 1 Abt. Orig. 1921. June 28. Vol 86. No. 5. pp. 396–405.

Professor Snapper concludes from his experiments :—

1. That "El Tor" vibrios can be found in stools of persons not suffering from true cholera.
2. "El Tor" vibrios give rise to haemolysis in an emulsion of goat's blood corpuscles; true cholera vibrios do not.

[It is generally recognized that the "El Tor" vibrio is a variety and near relative of the type cholera vibrio. That "El Tor" causes haemolysis is also well known.]

J. H. T. W.

Pergola (M.). **Valore dell'arbutina nell'identificazione dei vibrioni.** [Value of Arbutin in Identification of Vibrios.]—*Ann. d'Igiene.* 1921. May. Vol. 31. No. 5. pp. 265–271.

Using culture media of ordinary broth, to which arbutin was added in varying amounts, Professor Pergola arranges his vibrios according to their power of attacking the arbutin. Typical "commas" act strongly.

J. H. T. W.

Małecinski (J.) **Cholera azajatycka wśród ludności cywilnej miasta stolecz. Warszawy w roku 1920. (Sprawozdanie wydzialu zdrowia publicznego miasto stol. Warszawy.)—Cholera asiatique parmi la population civile à Varsovie en 1920. (Compte-rendu du Bureau municipal de Santé de le ville de Varsovie.)**—*Przegl. Epidemjol.* Warsaw. 1921. Vol. 1. No. 4. pp. 384–385. [French Summary p. 386.]

Merely notes occurrence of infection. Number of cases 22; deaths 15, or 68 per cent.; 5,705 persons were vaccinated.

J. H. T. W.

SCURVY.

BASSETT-SMITH (Percy W.). **Further Experiments on the Preservation of Lemon Juice and Prevention of Scurvy.**—*Lancet.* 1921. Aug. 13. pp. 321–322. With 2 charts in text.

Previous papers on this subject by Sir Percy Bassett-Smith were noticed in this *Bulletin*, Vol. 16, p. 402, and Vol. 17, p. 194; in the present paper the author records the results of testing his tablets after prolonged storage.

"I have therefore tested healthy young guinea-pigs of about 300 gm. weight with tablets [prepared from fresh lemon juice in vacuo without heat] which had been stored in the laboratory at room temperature for over twelve months. The basal diet consisted of ships' biscuit, bran, oats, casein, salt and lime-water, with 60 cc. daily of milk so heated as to destroy any antiscorbutic vitamin. Each day one-fifth of a tablet was given, equivalent to 4 cc. of fresh lemon juice. The animals under experiment have over a period of 70 days showed no signs of scurvy; they are bright and healthy, and have steadily gained weight, as shown by the curves in the annexed chart. Control animals, without antiscorbutic factors, show signs of scurvy about the twenty-first day and then rapidly lose weight."

It was shown in the last paper that short periods of heating, even at boiling temperature, only slightly reduce the antiscorbutic value of lemon-juice,* and it is suggested that the tablets could be produced in commerce by a process similar to that used in the preparation of Nestlé's condensed sweetened milk, which retains most of its vitamins.

A sample of lemon-juice extracted from the whole lemon at Messina was tested and found to be worthless when used in quantities up to 8 cc. daily; it had not been specially protected from heat due to climate and storage. The same was the case with a Messina sample preserved with potassium metabisulphate.

Tinned tomatoes were tried and were well taken by the guinea-pigs; the results of GIVEN and MCCLUGAGE, and of HESS, were confirmed [see this *Bulletin*, Vol. 18, p. 56]. It was effective in 4 cc. daily doses whether as prophylactic or after onset of scurvy symptoms. Charts illustrate the first two experiments.

A. G. B.

VEDDER (Edward B.). **The Etiology of Scurvy.**—*Milit. Surgeon.* 1921. Aug. Vol. 49. No. 2. pp. 133–150. With 9 charts.

The author draws attention to the variation in results caused by using different kinds of animals in experiments—rats being, for instance, little susceptible to scurvy; guinea-pigs much more so. He discusses the various terms used for the active preventive factors, and introduces the name "Vitaliment" as being more appropriate than "Vitamin," accessory food factor, or water soluble B and C and fat-soluble A. He describes a fresh series of experiments on guinea-pigs, the results of which are given in the following summary:—

"1. Scurvy can be produced in guinea-pigs with regularity by feeding a liberal and varied diet consisting of oats, mixed scratch feed, bread, sterilized milk, and hay.

* In the last sentence of the summary of Sir Percy Bassett-Smith's last contribution [Vol. 17, p. 194] the word *less* was unfortunately substituted for *more*.

" 2. Scurvy may be prevented by the daily addition of 5 gms. of green grass or 5 cc. of filtered orange, lemon, or grapefruit juice, and cured by larger amounts.

" 3. The antiscorbutic vitaliment is soluble in water, absolute alcohol, acetone, and commercial ethyl acetate.

" 4. The antiscorbutic vitaliment cannot be extracted from the partially dried juice by ether, chloroform or carbon tetrachloride.

" 5. The antiscorbutic vitaliment differs from the beriberi-preventing vitaliment in the following particulars :—

> " (*a*) The antineuritic vitaliment is adsorbed by finely powdered animal charcoal. The antiscorbutic vitaliment is not so adsorbed. (Harden and Zilva—confirmed by the writer.)
>
> " (*b*) The antineuritic vitaliment is precipitated by phosphotungstic acid. The antiscorbutic vitaliment is not so precipitated, but is probably destroyed.
>
> " (*c*) The antineuritic vitaliment is a nitrogenous base. The antiscorbutic vitaliment is apparently non-nitrogenous.

" 6. By means of the solvents mentioned above a purified extract has been prepared which is suitable for further chemical study."

P. W. Bassett-Smith.

i. Orenstein (A. J.). **The Etiology of Scurvy.**—*Proc. [Transvaal] Mine Med. Officers' Assoc.* 1921. Apr. Vol. 1. No. 1. pp. 2–6.
ii. Donaldson. **The Clinical Side of Scurvy in the Mine Native Labourer.**—*Ibid.* p. 6–9. (Discussion pp. 10–11.)

i. In 1920 the number of cases of scurvy diagnosed in the Rand Mines was greater than that of the total of the preceding five years. The various etiological theories are discussed, but nothing new is brought forward.

ii. The general clinical symptoms are described. Diagnosis was difficult in some of the more acute forms found in young natives, with extensive blood extravasations under the skin and into joints or serous cavities. The respiratory organs were less implicated than is usually the case in Europeans, and the central nervous system was rarely affected. Haematuria is common and periarticular extravasations are frequent, the latter cases often being sent to hospital as rheumatism. The late effects of scurvy, such as contractures and deformities, are now uncommon, as the cases generally come earlier under treatment. To show the prevalence of the disease it was stated that, in 1919–20, 200 cases of active scurvy were treated in one hospital, and one third of these occurred within the first three months of joining the mines. The importance of supplying the workers with a rational antiscurvy diet was evident, and oranges and lemons were recommended rather than a preparation called " Lemnos," which was often advocated. A supply of sour milk and sauerkraut would be useful. Dr. Watkins drew attention to the over-cooking of the meat ration, and stated that no vegetables should be boiled ; they *ought to be steamed*. The value of Kaffir beer was uncertain, as its preparation differed in different mine areas. There was no doubt that scurvy predisposed to every kind of bacterial invasion, and it was possible to show that the fall of pneumonia in the mines ran parallel with the fall in scurvy cases.

P. W. B-S.

Koch (Walter). **Der jetzige Stand der Pathologie und Pathogenese der Barlowschen Krankheit und des Skorbuts.** [The Present Position with regard to the Pathology and Pathogenesis of Barlow's Disease and Scurvy.]—*Deut. Med. Woch.* 1921. June 30. Vol. 47. No. 26. pp. 749–750.

The author has placed before the reader very clearly the subject treated, but gives no new facts. He considers that Barlow's disease and the scurvy of infants and adults are identical diseases, the manifestations of which change with the age of the patient. He describes the pathological characters which are found, particularly the haemorrhages, and the epiphysial changes in infants, to which are added in older children ulcerative stomatitis and carious teeth. Insufficient quantity of food leads to hunger oedema, whereas scurvy is produced by food defective in quality. Sucklings appear to be born with a store of vitamins, as scurvy does not occur before the age of six months. He states that though the various antiscorbutic foods act best when taken in the raw state, they do not lose much in strength by ordinary cooking, but are greatly reduced by long heating, drying, or prolonged storing. He condemns sterilized and prepared milk or artificial foods for sucklings. A food defective in quality predisposes to scurvy in proportion to the rate of growth in the children. Constitutional peculiarities and infective diseases predispose to and favour the onset of scurvy. Our present knowledge has rendered the diagnosis of the disease more easy, and no disease can now be more readily cured, though we have not yet discovered finally the etiological factors.

P. W. B-S.

Comby (J.). **Douze nouveaux cas de scorbut infantile.**—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1921. Mar 3. Vol. 45. No. 7. pp. 288–293. (Discussion, pp. 293–296.)

After drawing attention to the fact that owing to the shortage of fresh milk and ignorance or negligence of the causes, infantile scurvy has much increased in France, the author describes the clinical characters of two cases, and states that by simple antiscorbutic treatment every one of the 72 seen by him had been cured, even when of long duration and severe character. Painful pseudo-paraplegia was recognized in 68 cases, haemorrhagic gums in 16 (22 per cent.), seven times in infants who had not erupted any teeth. When the deficiency food was replaced by freshly boiled milk, with orange juice or sweetened lemon juice, rapid improvement was generally noticed, and for prophylaxis, even with the use of sterilized, condensed or dry milk, the addition of fresh juice was efficacious.

In the discussion, M. Variot condemned artificial infant milk foods, but said that with sweetened condensed milks he had very good results, in great contrast to those following the use of unsweetened milk. From clinical observations he found the effect of "dried" milk on infants was deplorable; it was badly tolerated and badly utilized, and often gave rise to gastro-intestinal trouble; though perhaps useful for adults, it was not a good food for infants. Louis Martin and the author spoke favourably of dried milk, but no preserved milk could replace efficiently raw cows' or mothers' milk.

P. W. B-S.

FABER (Harold K.). **Infantile Scurvy following the Use of Raw Certified Milk.**—*Amer. Jl. Diseases of Children.* Chicago. 1921. Apl. Vol. 21. No. 4. pp. 401–405.

It is stated that infants may develop scurvy when brought up on a diet of raw milk. The author reports the case of a child which had been fed exclusively on certified raw milk and at the age of six months developed typical scurvy. It recovered completely when a diet rich in antiscorbutics was supplied. It is possible that the amount of milk given was too small, or that something was added which destroyed the antiscorbutic present in the milk, for example, sodium citrate, which had been given in small quantities. Though this theory was not conclusively proved, the author suggests that the use of sodium citrate in infant feeding over long periods should be provisionally regarded as dangerous.

P. W. B-S.

MCCLELLAND (R. S.). **A Case of Scurvy.**—*Lancet.* 1921. Sept. 17. p. 608.

The case reported was that of a collier, aged 40, from North Wales, who showed typical symptoms of scurvy. It was disclosed that he lived almost entirely on bacon, bread and tea, and disliked vegetables, which he called pig's food.

P. W. B-S.

MACKLIN (A. H.) & HUSSEY (L. D. A.). **Scurvy: a System of Prevention for a Polar Expedition based on Present-day Knowledge.** —*Lancet.* 1921. Aug. 13. pp. 322–326.

An interesting and carefully thought-out description of the practical means of defending an expeditionary party from scurvy in Polar regions.

P. W. B-S.

GODLEWSKI (Henri). **Carence partielle et préscorbut.**—*Presse Méd.* 1921. Aug. 27. No. 69. pp. 682–683.

The author states that acute cases of scurvy are rare in comparison with those grouped under the name of pre-scurvy anaemia, which condition is caused by a partial deficiency in the food supplied, and easily passes into confirmed scurvy if the dietetic error is not corrected. This condition of chronic scurvy, as described by HOLST and FROLICH, WEILL and MOURIQUAND, is compatible with a state of health generally satisfactory but very unstable. In the scorbutic anaemia, with the development of the osseous changes the number of red cells becomes reduced as low as 2,500,000 per cmm. and normoblasts appear. This is a condition frequently found in infants badly dieted, who have intestinal symptoms commonly ascribed to teething, and a further stage is Barlow's disease. The author emphasizes the fact that for cure it is necessary to give, not traces, but full doses of the vitamin-containing substance both to animals and man. In children the loss of vitamin has been of long duration, and marked changes of metabolic activity have been induced, so that an acute condition is readily brought about by accidental causes. "Traces of the living substance in the food of the infant serve to keep it alive, but in a state of anaemia and pre-scurvy. For a normal growing state it is necessary that not only

traces, but a daily ration of the essential substance be given, which is probably required for nuclear formation." For treatment thyroid extract, fresh vegetables, "eau de vegetation," milk, fresh fruits and meat are useful. He quotes TROUSSEAU, who gave his little son, aged six months, 500 gms. of pounded meat every day for scurvy diarrhoea, with success; the author recommends about 25 gms. under the same conditions.

P. W. B-S.

GERSTENBERGER (H. J.). **Malt Soup Extract as an Anti-Scorbutic.**—*Amer. Jl. Diseases of Children.* Chicago. 1921. Apr. Vol. 21. No. 4. pp. 315–326.

It has generally been considered, and HESS has reported, that KELLER's malt soup—a mixture which contains 100 gms. of so-called malt soup extract—is a diet that induces scurvy. For certain reasons infants were fed on this preparation, and instead of getting worse, they showed marked evidences of improvement. This was attributed to the brand used having been made from barley of the proper age and state of germination, containing a high amount of potential antiscorbutic material. The author suggests that all malt soup extract could be so manufactured as to ensure a large antiscorbutic value, in which case it could be added to the list of regular and trustworthy antiscorbutics. Three other cases were, however, later treated with the same brand, with less satisfactory results.

P. W. B-S.

FINDLAY (G. Marshall). **A Note on Experimental Scurvy in the Rabbit and on the Effects of Ante-Natal Nutrition.**—*Jl. Path. & Bact.* 1921. Oct. Vol. 24. No. 4. pp. 454–455. With 1 plate.

The animals that have shown susceptibility to experimental scurvy are monkeys, pigs and guinea-pigs. The author found that rabbits, too, were, under certain conditions, affected with symptoms and signs similar to those in guinea-pigs. Six rabbits, three males and three females, were kept on a diet of oats, wheat-bran and water. Two of these crossed during the experiment gave birth to seven young, which were all born dead and showed signs of scurvy, namely, swollen knee joints, haemorrhage in the tissues round the joints, enlarged costo-chondral junctions, etc. No signs of scurvy were found in the adult animals even up to the sixty-third day, though there was considerable loss of weight; when placed on swede juice the animals gained weight and improved in condition. The author concludes that (1) rabbits deprived of vitamin C gradually lose weight and eventually die; (2) the addition of vitamin C brings about a cure; (3) pregnant rabbits deprived of vitamin C throughout the period of gestation produce young with marked scurvy signs, the mothers remaining apparently healthy.

P. W. B-S.

FINDLAY (George Marshall). **The Effects of an Unbalanced Diet in the Production of Guinea-Pig Scurvy.**—*Biochemical Jl.* 1921. Vol. 15. No. 3. pp. 355–357.

The experiments were instituted to see if an excess of sugars, fats or proteins in the diets of guinea-pigs deprived of antiscorbutic

vitamin would hasten the onset of the disease, as has been found in the case of vitamin B deficiency. Twenty-four animals were used, in four groups of six in each, and tables of weights, etc. are given. It is specially noted that the biological appearances found in the bones and joints of the animals which received the extra ration of cod-liver oil were identical with those met with in other cases of guinea-pig scurvy, showing that the want of vitamin A is in no way responsible for the lesions which occur in experimental scurvy. From the experiments they conclude that an excess of either fat, protein or carbohydrate in a diet deficient in vitamin C does not, in guinea-pigs, hasten the onset of the symptoms, but that death is slightly accelerated when there is an excess of protein or carbohydrate, and the loss of weight is greatly increased in scurvy guinea-pigs when there is an excess of carbohydrate.

P. W. B-S.

Tozer (Frances M.). **The Effect on the Guinea-Pig of Deprivation of Vitamin A and of the Anti-Scorbutic Factor, with Special Reference to the Condition of the Costo-Chondral Junctions of the Ribs.**—*Jl. Path. & Bact.* 1921. July. Vol. 24. No. 3. pp. 306–325. With 2 plates, 5 text figs. and 2 charts.

The histological changes in the guinea-pigs used in the prolonged experimental work carried out at the Lister Institute with regard to scurvy, some of which have been already published, are here described in detail. The author points out that in the early experiments by Holst, and at the Lister Institute, the importance of vitamin A was not fully appreciated, and its absence caused erroneous conclusions to be drawn. The experiments here detailed are divided into four groups :—

1. Normal controls, namely, animals fed on the basal diet, plus vitamin A, B and C.
2. Animals deprived of antiscorbutic factor only.
3. Animals deprived of A only.
4. Animals deprived of A and C vitamin.

The changes of the costo-chondral joints under each condition are described and figured, and the general course of the illness produced is clearly described. In No. 2 the haemorrhages and disorganization of the joints are very rapid and marked, and death occurs within a month. In No. 3 the course is not so rapid, haemorrhages are not present, the joint degeneration is less marked, but medullary changes are more in evidence, and death is less rapid. In No. 4 the appearances were much the same as those of scurvy alone. These experiments show that unless the animal is protected against the effects of insufficient vitamin A, the results will be complicated and the diagnosis may be incorrect. The clinical picture in guinea-pigs deprived of the antiscorbutic factor alone is graver than when vitamin A alone is lacking. Simultaneous deprivation of both these accessory factors does not seem to accelerate the onset of scurvy symptoms, but after these appear they mask the less severe symptoms caused by the absence of vitamin A, so that the disease appears to be scurvy. Histological appearances at the costo-chondral junction resembling

those of rickets have been observed in animals deprived of vitamin A, and in mild and acute scurvy complicated by a deficiency of vitamin A. The two beautiful plates show clearly the conditions produced.

P. W. B-S.

Morel (A.), Mouriquand (G.), Michel (P.) & Thévenon (L.). **Sur l'absence de troubles électifs du métabolisme du calcium osseux dans le scorbut expérimental.**—*C.R. Soc. Biol.* 1921. July 23. Vol. 85. No. 27. pp. 469–470.

The remarkable changes in the bones of guinea-pigs suffering from experimental scurvy led the authors to examine for marked evidences of decalcification and loss of salts. They found in normal animals for 100 parts of dry bone 56·37 ash and 25·9 lime, in animals with clinical and anatomical scurvy 58·7 ash and 26·9 lime, in animals which died of intestinal intoxication without scurvy lesions 59·21 ash and 26·8 lime; in animals which died from simple inanition the ash was 51·4 and the lime 25·7. These results indicate that the alteration of the bones in experimental scurvy is not due to the loss of mineral material and especially lime. Further work is being carried out, including histological examinations. [These have been described and well figured by Tozer of the Lister Institute, London: see above.]

P. W. B-S.

Findlay (G. Marshall). **The Blood and Blood-Vessels in Guinea-Pig Scurvy.**—*Jl. Path. & Bact.* 1921. Oct. Vol. 24. No. 4. pp. 446–453.

In this investigation particular attention was directed to the changes in the blood and vascular system. Thirty guinea-pigs of 250 to 400 gms. were used: 25 were placed on a diet of bran and oats with 60 cc. of autoclaved milk, with the addition in the case of the five controls of 5 cc. of fresh orange juice. Twelve of the experimental animals were killed at intervals of 48 hours, commencing on the second day, while 12 were allowed to develop well-marked scurvy symptoms and were then killed—six in the pre-coma and six in the coma stage. Cultures from the heart blood were negative except in one case. Blood counts from the ear and heart blood taken at the same time [it is not stated whether this was before or after death] showed considerable variations demonstrating marked capillary stasis something like that seen in cases of shock; eventually the stagnation leads to insufficient oxygenation of the tissues and then death. No marked erythroblastic reaction was found in the bone marrow. The chief pathological changes in the vascular system were:—(1) Degenerative changes in the lining endothelium; (2) Extreme congestion; (3) Fine oedema of the tissues around the vessels; (4) Haemorrhagic areas in close relationship to the capillaries. Fatty degeneration of the heart muscles was not found, but haemorrhagic infiltration of the heart was seen in five cases. The earliest haemorrhage was found microscopically after 12 days in the muscles of the back of both knees; by the 14th day they were visible to the naked eye. The internal organs showed haemorrhagic foci most commonly in the kidney, bladder, intestine, liver, adrenals, bone marrow, and spleen; in some of the animals at the time of death the haemorrhages were very small and few in number. Haemorrhage into the cortex of the adrenals was found only in four animals but subcapsular haemorrhages were common.

In adult animals haemorrhages into the central nervous system were never observed, though they were frequent in the meninges. In guinea-pigs the disease is acute from a total deprivation of the vitamin C, whereas in man the deficiency is very rarely complete and the disease is much more chronic [as has lately been emphasized by many observers]. In one instance the guinea-pig when killed had in utero four young between 50–60 days old; these all showed well-marked scurvy lesions, and in one there were extensive haemorrhages in the central nervous system.

P. W. B-S.

Hess (Alfred F.), Unger (L. J.) & Supplee (G. C.). **Relation of Fodder to the Anti-Scorbutic Potency and Salt Content of Milk.**—Reprinted from *Jl. Biol. Chem.* 1920. Dec. Vol. 45. No. 1. pp. 229–235. With 2 charts in text.

Five cows were fed for 21 days upon an ample diet, except that it was deficient in antiscorbutic vitamin. Samples of the milk, which had fallen off in quantity, were taken for further examination, the cows were then kept for three weeks on pasture, and samples were again taken from the five. The samples were dried by the Just Hatmaker process and guinea-pigs were simultaneously fed with the two kinds, using a daily ration equal to 80 cc. of the fresh milk. Those fed on the dry fodder milk developed scurvy in about 21 days, and died within 65 days; the animals on the pasture milk remained alive up to the 120th day, though two showed slight scurvy signs [number of guinea-pigs used not stated]. It would appear that the antiscorbutic taken in with the food is rapidly secreted in the milk, and that no storage in the body takes place, also that dry fodder contains but little antiscorbutic vitamin. A chemical examination of the milk showed that in the two samples the fats, protein, and sugar percentages were very similar, but that the calcium, phosphate, and citric acid content was much higher in the pasture milk than in the dry fodder milk.

P. W. B-S.

Mouriquand (G.) & Michel (P). **Le jus de citron stérilisé est-il anti-scorbutique ?**—*C.R. Soc. Biol.* 1921. July 23. Vol. 85. No. 27. pp. 470–472.

Holst and Frölich found that lemon-juice heated to 110° C. did not lose its antiscorbutic power, being stabilized by the presence of citric acid. The authors found, in their experiments, that the juice heated for an hour at 120° C. when given to guinea-pigs in doses of 5–10 cc. did not prevent scurvy, but delayed its onset for a period of 85–110 days, causing a chronic and peculiar type of disease, which underwent, sometimes, spontaneous cure [see this *Bulletin*, Vol. 18, p. 60.] From their experience they conclude that sterilization by heat does reduce the antiscorbutic power of the juice. The experimental scurvy thus produced only appears after a prolonged period and more nearly resembles the human form; it is necessary to watch the animals for a period of 150 days, the failure to do which has led many experimenters to erroneous conclusions, and explains some discordant results. [This applies to conditions of hypo-vitaminosis, not to experiments where the vitamin is completely withheld.]

P. W. B-S.

i. Ellis (N. R.), Steenbock (H.) & Hart (E. B.). **The Stability of Anti-Scorbutic Vitamine and its Behaviour to Various Treatments.**—*Jl. Biol. Chem.* 1921. Apr. Vol. 46. No. 2. pp. 367–380.

ii. Hart (E. B.), Steenbock (H.) & Ellis (N. R.). **Anti-Scorbutic Potency of Milk Powders.**—*Ibid.* p. 309.

i. In continuation of previous experiments the authors working with guinea-pigs found that desiccation of cabbage in an atmosphere of CO_2 for 36 hours at 65° C. did not prevent the destruction of the antiscorbutic vitamin. This is contrary to the results reported by Zilva when boiling lemon-juice in an atmosphere of CO_2 [this *Bulletin*, Vol. 18, p. 60]. They found that in the making of sauerkraut from cabbage, and silage from corn, fermentation processes caused a destruction of the antiscorbutic factor. The vitamin was not removed from orange-juice by ether or by aeration, but was destroyed by oxidizing agents such as hydrogen peroxide and potassium permanganate. The reducing action of molecular hydrogen had no effect, but a certain amount was lost by passage through blood charcoal or a Chamberlain filter. Thirteen illustrative charts are given of the weight curves.

ii. Three methods of drying milk are commonly used in America: (1) the Merrell-Soule or spray process; (2) a spray process used in the California Central Creameries, in which the milk is not condensed before being dried; and (3) the Just process of drying the milk on heated rollers. These products were used for the experiments, guinea-pigs being employed. Descriptive weight charts of the animals are given. It is recognized that milk powders may vary in their antiscorbutic properties, depending upon, firstly, the initial quantity of the vitamin in the milk, which is influenced by the feeding of the cows as reported by Hess, Unger, Supplee and others; secondly, upon the method employed in drying, the spray process being more destructive to the antiscorbutic vitamin than the Just process. They point out that this does not condemn the milk powders as food, but it should be recognized that when used as the sole source for infant feeding they must be supplemented by some substance containing the necessary vitamin, the only possible exception being powders prepared by the Just process from summer milk, or from cows especially fed.

P. W. B-S.

Zilva (Sylvester Solomon) & Miura (Masataro). **The Differential Dialysis of the Antineuritic and Antiscorbutic Factors.**—*Biochemical Jl.* 1921. Vol. 15. No. 3. pp. 422–426. With 2 charts.

It has been shown by Eijkman, Holst, and Frölich that the accessory food factors for beriberi and scurvy are dialysable. The authors prepared collodion thimbles, and their permeability was determined by means of chemicals, mostly dyes. Filtered autolysed yeast was used for the antineuritic factor, and de-acidified filtered lemon-juice for the antiscorbutic. The actions were similar, both practically failed to pass through the thimbles which had been soaked for 24 hours in 90 per cent. alcohol, but completely through the 95 per cent. one. A rat was used for the beriberi, and a guinea-pig

for the scurvy experiments. The permeability of the colloidal membrane that allowed the diffusion of the "factors" was the same as that which permitted the passage of methylene blue, neutral red, and safranine. It is suggested that the active molecules of the factors may be of a semi-colloid nature and further investigations are suggested.

[It is rather remarkable that the guinea-pig on 7 cc. of dialysed lemon-juice should show scurvy symptoms in so short a time as five days, seeing that an observation-period so much longer is required for symptoms in the other animals.]

P. W. B-S.

DIETETIC DEFICIENCY DISEASE.

HOPKINS (F. Gowland). **The Huxley Lecture on Recent Advances in Science in their Relation to Practical Medicine and the Nutritional Requirements of the Body. Delivered at Charing Cross Hospital Medical School (University of London), on November 24th, 1920.**—*Lancet.* 1921. Jan. 1. pp. 1–7.

Professor Hopkins begins his remarks with a discussion of what may be termed "the other side." He pays high tribute to the great workers in the science of Calorimetry, but adds that results obtained from this science are insufficient in practical dietetics. In this connection Professor Hopkins gives the "text" of his lecture. "An animal may receive, digest, absorb, and, in a sense, fully metabolize a diet consisting of all the recognized basal foodstuffs. It may be taking these foodstuffs in quantities sufficient, or more than sufficient, to supply all its needs for energy. And yet its nutrition may be so abnormal that neither growth, nor health, nor life, is long maintained." The chemical details of a diet just as much as its quantity and energy content make it satisfactory.

Contrary to this view is RUBNER'S law of Isodynamic Equivalence: "This involves the principle that any one foodstuff can replace any other so long as the replacement makes no change in the supply of energy."

Professor Hopkins points out that statements like this if not used with great care may become the basis for false doctrine. RUBNER also states that "the duration of growth and the ultimate size of any animal are both functions of the rate of energy exchange in its tissues." Thus it appears that the mouse is small and the elephant large.

The work of VON PIRQUET is next discussed. This worker has applied strict scientific principles to human alimentation. This system, known as the "Nem" system, takes for its unit the energy derived from 1 cc. of milk. ("Nem" is the name given to this unit.) The Nem system is applied to nearly 200,000 school children in Vienna on behalf of the Red Cross Organization. In short, the diet is reckoned in so much energy, 10 per cent. of which is derived from protein. If fat is scarce, sugar is substituted, and so on; so long as the energy content of the food is maintained the ingredients do not matter. This position also is in opposition to Professor Hopkins and his school. The experiments of KROGH and LINHARD of Copenhagen are quoted to show that there is a limit to the interchangeability between fat and carbohydrate.

The rôle of protein in diet receives the consideration it deserves, and certain of the facts regarding the nature and synthesis of protein are discussed. The vitamin question follows logically and the lecturer shows how the "law of the minimum" is more important than the law of Isodynamic Equivalence. It is admitted that when "a diet is harmoniously balanced in a chemical sense, then indeed energy does become the sole limiting factor." In fact, there is a minimum amount of all the factors concerned in dietetics below which the diet fails.

Scurvy, both in its history and modern treatment, is discussed and stress laid upon its production by deficiency. Fat-soluble vitamin, it is stated, will play an important part in the future. Pellagra is

linked up with the food deficiency diseases, and the work of both GOLDBERGER and WILSON is described in this connection.

The concluding remarks concern the visit of Dr. Harriette CHICK and Dr. Elsie DALYELL to Vienna, under the auspices of the Lister Institute. These workers have gone to the home of the Nem system and are applying the newer ideas of dietetics in the wards of VON PIRQUET. "In adjacent wards the Nem system will go on undisturbed. A real comparison will therefore be made."

No prophecy is made concerning the outcome of this experiment, but undoubtedly the results will be of enormous importance to the science of nutrition and therefore to practical medicine.

A. D. Bigland.

HESS (Alfred F.). **Newer Aspects of Some Nutritional Disorders.**—*Jl. Amer. Med. Assoc.* 1921. Mar. 12. Vol. 76. No. 11. pp. 693–700. With 2 charts in text.

This contribution takes the form of a paper read before the Harvey Society on 15th January, 1921. The opening remarks concern the history of the subject, and tell of the remarkable advances during recent years. A warning is given to those who regard vitamins merely as substances without which certain specific diseases, as beriberi and scurvy, occur. The subject is more far-reaching than this, since between perfect health and these well-recognized deficiency diseases there is a wide area where flourish obscure alterations in nutrition and "ill-defined functional disabilities," due to the lacking of certain diet constituents. "It is such incomplete larval forms of the deficiency disorders to which physicians will have to address themselves." Not in man alone but also in the animal kingdom are food deficiency diseases to be found.

Scurvy.—The subacute and latent forms of this disease both in the infant and the adult are discussed, and attention is drawn to the great importance of recognizing types far more prevalent than the text-book variety. The disease shows itself in three particular ways:—(1) A lesion of the lining of the blood vessels, which allows blood to escape and gives rise to all the haemorrhagic conditions associated with the disease. The blood as regards its coagulability is practically normal. A tourniquet applied to the upper arm in these cases will usually cause the appearance of numerous petechiae on the forearm. This condition is not found in any other of the food deficiency diseases, and demonstrates the weakness of the vascular wall. (2) A lesion of the bones. (3) Circulatory disturbances. These furnish a link between scurvy and beriberi. Tachycardia (140–150 in an infant) and marked instability of the pulse-rate are early and characteristic signs. Accompanying these phenomena is a polypnoea (40, 50, or 60 per min.). The complex is known as the "cardio-respiratory phenomena" of scurvy. It yields promptly to antiscorbutic treatment. Enlargement of the heart, especially the right side, and oliguria are also characteristic signs.

As regards the source of antiscorbutic vitamins in temperate zones the potato is all important, and among animal foods milk, of course, is a basal diet for infants. Vegetables must be in good condition or vitamin yield may be lacking, and in the case of milk everything depends upon the fodder of the cow. Many factors causing destruction of the antiscorbutic vitamin are discussed, and stress is laid upon

duration of heating rather than the actual degree of temperature reached, and the great importance of oxidation.

Rickets.—The author thinks that rickets is primarily a dietetic disorder, but that hygienic factors are important contributary influences.

The author concludes his paper with a reference to the inter-relationship of nutrition and infection.

A. D. B.

McCarrison (Robert). **The Influence of Deficient and Ill-balanced Dietaries in Favouring the Development of Gastro-Intestinal Infections.**—*Indian Jl. Med. Res.* (Special Indian Science Congress Number, 1920.) pp. 38–43.

Two theses are propounded :—(1) " That the freedom of the gastro-intestinal tract from infection depends in the main on the health of the gastro-intestinal mucosa." (2) " Imperfect, ill-balanced and insufficient food is the most ready means of inducing an unhealthy state of the gastro-intestinal tract."

A list of the functions that a good food must subserve is enumerated, and the factors which limit the development of gastro-intestinal infections are discussed. The common factor of all the protective sources is " perfect food." As evidence of the effect of diet upon the intestinal mucosa the author recapitulates his experiments upon monkeys, from which two important facts emerged : (1) " That insufficiency of vitamins is a potent source of bowel complaints " ; and (2) " That deficiency or excess of proximate principles in the food greatly favours the onset of gastro-intestinal disorders." The naked eye appearances of the intestines of these monkeys have been already described in this *Bulletin* [Vol. 16, p. 79]. Microscopically, micro-organisms were found to have penetrated even as far as the serous coat of the intestine.

The author does not wish it to be understood that changes of such severity occur often in human subjects, but undoubtedly these results do show what deficient diet carried to its extreme can do. To prove that somewhat similar changes, though less in degree, may occur in human subjects, a case is described of a man who for twenty years had been a confirmed invalid with obstinate constipation, dyspepsia and dilated stomach. An analysis of this man's diet and suitable correction thereof brought about a cure in a short time. One of the factors introduced into the curative diet of this case was a vitamin extract made from the yolk of eggs by alcoholic extraction. Now when this extract was discontinued the patient noticed that his stools which had become dark coloured resumed their white, offensive condition. It is assumed that the vitamin extract promoted the pancreatic and biliary flow. In confirmation of this is cited the work of Voegtlin and Meyer, who showed that intravenous injections of extract of brewer's yeast stimulated the biliary and pancreatic secretions, as does secretin.

The paper concludes with an account of Besredka's experiments at the Pasteur Institute of Paris. Ox-bile is given to healthy rabbits ; a few hours later an emulsion of typhoid or paratyphoid bacilli is administered orally. Typhoid or paratyphoid supervene. Healthy rabbits to which typhoid emulsion is administered without a previous dose of ox-bile do not develop the disease. Besredka stated that the

action of ox-bile was to cause intestinal erosions, and thus allow the bacilli to gain a footing. The author repeated these experiments with a single rabbit and its control, and obtained the same results.

A. D. B.

Fabroni (Bruno). **Sindromi fruste od associate nelle distrofia alimentare per avitaminosi.**—*Riv. Pellagrolog. Ital.* 1920. Sept.–Dec. Vol. 20. No. 5–6. pp. 35–41.

The writer has observed in 42 patients, treated in one of the war-stricken districts of Italy between the end of 1918 and the summer of 1920, what he describes as abortive syndromes of avitaminosis (*sindromi fruste d'avitaminosi*). After a longer or shorter period of general debility with anorexia and transitory headaches, the patient experiences an afebrile disorder of the gastro-enteric apparatus. A diarrhoea appears, which is rebellious to every astringent treatment, and is accompanied by nausea and distaste for the habitual food. At the same time there is oliguria and slight oedema, which comes and goes without apparent cause. The patient is easily fatigued, and complains of dyspnoea and tachycardia. Sometimes there is a sensation of dryness in the fauces and throat, followed, after some days, by gingivitis. In other cases there may be slight ecchymoses or definite petechiae. Myalgia, disorders of the sense of touch and increased reflexes are also observed.

In addition to the cases grouped under this syndrome, the writer discusses three "mixed" groups. In the first, comprising eight cases, the symptoms included some of the typical symptoms of scurvy. In the second group, of five cases, the marked development of dropsy was the most notable of some symptoms suggesting "hunger oedema." In the third group (10 cases) the symptoms of pellagra were the most prevalent.

J. Rosslyn Earp.

Cramer (W.), Drew (A. H.) & Mottram (J. C.). **Similarity of Effects Produced by Absence of Vitamins and by Exposure to X-rays and Radium.**—*Lancet.* 1921. May 7. pp. 963–964.

The authors state that "vitamins are necessary for the maintenance of life of a highly differentiated animal as a whole, but not necessarily or probably of its individual cells." Lymphoid tissue, however, is one tissue which is probably affected in avitaminosis. In mice and rats fed upon a diet containing no vitamins the spleen is found to be much wasted, the thymus (a large organ in these animals) is hardly visible, and the Peyer's patches are difficult to recognize with the naked eye. Microscopically, the spleen and lymph glands are found to be almost bare of lymphocytes, while blood-counts show a reduction in the number of lymphocytes and an increase in the number of polymorphs. "This condition of the lymphoid tissue is practically identical with that produced by exposure to X-rays and radium." Not only is the lymphoid tissue affected, but the animals die in a state of emaciation. The authors suggest that these observations show that lymphoid tissue plays a more important rôle in nutrition than is usually recognized. Further investigations are proposed.

A. D. B.

PERROT (E.) & LECOQ (R.). **Sur la valeur alimentaire de quelques farines composées du commerce au point de vue leur constitution chimique et de leur teneur en vitamines.**—*C. R. Soc. Biol.* 1921. Mar. 19. Vol. 84. No. 11. pp. 529–530.

The authors recount their experiments on more than 60 animals with regard to the dietetic value of certain commercial flours. Twenty-three flours were thus examined. The white rat was the animal chosen for the work. With "diète hydrique" loss of weight is rapid and death occurs in less than fifteen days. Fed upon "pain actuel" the animals show at the beginning a certain increase in weight, followed by a long "plateau"; death is delayed, and in one case the rat lived for more than six months. Of the 23 commercial products, 17 showed a value lying between the figures derived from these two type diets. Some of these flours gave results very little different from those obtained with the "diète hydrique," and were followed by death almost as rapidly. Three flours were comparable to the bread regime, and three only were proved superior to this.

The authors draw attention to the gravity of the position thus revealed, especially as these commercial flours are intended for children and convalescents. They claim that we have the right to insist that the manufacturers reconstruct their manifestly insufficient formulae.

A. D. B.

SHORTEN (J. A.). **The Vitamin Content of Certain Sun-dried Vegetables.** —*Proc. Roy. Soc. Med.* (Sect. of Therap. & Pharm.). 1921. June. Vol. 14. No. 8. pp. 20–21.

A new and important industry has sprung up in Quetta concerned with the drying of vegetables in the air. The climate is such that "most vegetables can be completely desiccated in less than a day." The preliminary preparation includes cleaning, slicing, and scalding or steaming for a short time. The vegetables are then cut into slices and dried in the sun. They are finally compressed and placed in air-tight tins. The product apparently retains its original colour and taste even when cooked after a preliminary soaking in water.

It was decided to test these dried products with regard to their antiscorbutic and antineuritic vitamin content. For the former purpose guinea-pigs were used, while for the latter fowls were chosen. The animals were fed upon the usual basal diets, with the addition of a certain amount of the dried vegetable in question. Controls were used. It was found that as regards antiscorbutic properties, tomatoes, cabbages and potatoes, in this order, functioned well. Dried carrots, brinjal, spinach, turnip tops and turnips, together with a mixed dry vegetable product from Australia, showed little or no antiscorbutic properties. None of the sun-dried vegetables showed loss of antineuritic substance.

Animals fed upon the sun-dried products soaked in water, but uncooked, lived longer than those fed upon soaked and cooked samples. It is recommended that the products should be eaten in salad form.

Further experiments are in progress concerning the influence of the initial scalding and steaming process on the vitamin content.

A. D. B.

i. Hopkins (Frederick Gowland). **Notes on the Vitamine Content of Milk.**—*Biochemical Jl.* 1920. Dec. Vol. 14. No. 6. pp. 721–724. With 4 charts.

ii. Hawk (Philip B.), Smith (Clarence A.) & Bergeim (Olaf). **The Addition of Yeast to a Milk Diet.**—*Proc. Soc. Experim. Biol. & Med.* 1921. Mar. 16. Vol. 18. No. 6. p. 168.

iii. Rosenau (M. J.). **Vitamins in Milk.**—*Boston Med. & Surg. Jl.* 1921. May 5. Vol. 184. No. 18. pp. 455–458.

iv. Le Noir (P.) & Richet (C.). **Deficiency Disease.**—*Paris Méd.* 1921. May 7. Vol. 11. No. 19. p. 376.

i. In 1912 Hopkins first showed the startlingly favourable results following the addition of minute amounts of milk to synthetic diets incapable themselves of maintaining the growth and health of rats. Osborne and Mendel in 1920 reported that they were unable to confirm these results. Hopkins, in trying to repeat his earlier experiments, was successful with "summer" milk but failed with "winter" milk. Osborne and Mendel were unable to confirm this. A possible explanation of these disagreements is that there is a seasonal factor in the growth energy of rats.

ii. Hawk and his colleagues fed rats upon pasteurized milk and found that the addition of yeast to the milk increased the rate of growth. From this result, coupled with the fact that milk is poor in water-soluble B vitamin, the authors draw the conclusion that yeast may be found to be an important adjunct in baby feeding.

iii. Discussing canned and dried milks, Rosenau states that anti-scorbutic vitamin is the only one which may be affected in these preparations. The amount of this vitamin depends upon the content of the original milk and also upon the processes of heating and evaporation with special reference to oxidation.

iv. Le Noir and Richet are in complete agreement with the above statements.

A. D. B.

i. Bloch (C. E.). **Clinical Investigations of Xerophthalmia and Dystrophy in Infants and Young Children. (Xerophthalmia et Dystrophia Alipogenetica.)**—*Jl. of Hyg.* 1921. Jan. Vol. 19. No. 3. pp. 283–304. With 3 plates and 5 charts.

ii. Guerrero (L. E.) & Concepcion (I.). **Xerophthalmia in Fowls fed on Polished Rice and its Clinical Importance.**—*Philippine Jl. Sci.* 1920. July. Vol. 17. No. 1. pp. 99–103.

i. Children fed upon a diet deficient in "fat-soluble A bodies" develop dystrophia alipogenetica. This condition involves a lowered resistance to infection, and often leads to xerosis of the conjunctivae and cornea, with night blindness. Keratomalacia may result from this. In the first stages of the disease slight conjunctivitis with photophobia will be noticed. The disease generally occurs in the spring and amongst the children of the poorest country folk. The best treatment is cod-liver oil, or other substances containing fat-soluble bodies.

ii. The second paper shows that pigeons fed upon polished rice and "Tikitiki extract" may develop not only polyneuritis, but also xerophthalmia. The authors conclude that polished rice is therefore lacking in both vitamins A and B.

A. D. B.

KELLAWAY (C. H.). **The Effect of Certain Dietary Deficiencies on the Suprarenal Glands.**—*Proc. Roy. Soc.* Ser. B. Vol. 92. No. B 642. pp. 6–27. With 1 text fig. and 1 plate.

The author confirms the experiments of MCCARRISON with regard to suprarenal enlargement and increased store of adrenalin in pigeons fed upon polished rice. These changes are prevented by the addition of yeast extract. The increased adrenalin content is attributed to the diminished output of this substance as a result of lowered body metabolism. The oedema occurring in some cases of experimental neuritis is not due to increased output of adrenalin. The enlargement of the suprarenals is due partly to congestion and oedema of the glands, and partly to the storage of lipoid substances resulting from the breaking down of body tissues. In these pigeons the cholesterol content of the suprarenals is normal, though there is a well-marked hypercholesteraemia.

A. D. B.

TUBERCULOSIS IN THE TROPICS.

Scott (Henry Harold). **Report on Tuberculosis in the Far East: An Analysis of the Post Mortem Findings in 300 Consecutive Cases of Death from Tuberculosis, with Special Reference to the Disease as met with in Children in Hongkong.** [MS. Report dated 8th June, 1921, received from the Colonial Office.]

This Report deals with a long series of careful post-mortem examinations on patients, mostly children, dying from tuberculosis in Hong Kong. It is obvious that the investigation has been carried out on systematic lines by one well qualified for the work. The material at the author's disposal was large in amount and the results have been carefully analysed. The result is a report which is of a very high order of merit and calculated to add considerably to the existing knowledge as to the portals of entry of the tubercle bacillus.

The work is divided into four portions. In the first, the author discusses the epidemiology of the disease in general, the conclusions being drawn "firstly, that hereditary transmission of the bacillus is so rare that for practical purposes it may be declared negligible; secondly, that the incidence of tuberculosis depends on two main factors, namely, the dose received and the virulence of the strain inoculated, in other words, the degree of exposure to infection and the resistance which the inoculated subject is able to put forward, this, in turn, being dependent largely on his environment; thirdly, that there is not sufficient evidence, at present at all events, to afford support to the theory that there is such a thing as inherited predisposition to tuberculosis." The author is alive to the fact of acquired resistance as he mentions it specifically later on, quoting the experiments of Bryant in the immunization of guinea-pigs with small doses, but he does not seem to think it plays much part in Hong Kong. On the other hand, he sees "no reason for regarding the Chinese as exhibiting any special susceptibility to the bacillus of tuberculosis, in spite of the frequency with which cases are met with in the mortuary. The prevalence is in part due to the exclusion of light and air from their dwellings, but in large measure to the pernicious habit of spitting." He gives a very interesting picture of the housing conditions in Hong Kong, and these are undoubtedly such as greatly to facilitate the transmission of the bacillus from man to man. Without in the least desiring to minimize the importance of this bad environment, we would point out that the men of the Chinese Labour Corps in France, placed under relatively excellent conditions of housing and food, showed a very high incidence and mortality from tuberculosis in comparison to the British and French troops engaged in the same areas. From observations made during the war, we are inclined to impute to the adult Chinese a very definite degree of susceptibility or rather, as we prefer to put it, a very low degree of acquired resistance. In this connexion it is of great interest to note in the Report under review that "the ingestion of tuberculous milk, which is supposed to play a large part in the production of tuberculosis, especially intestinal tuberculosis, in children in England, has no influence here, for the Chinese children do not drink milk, nor does it arise here from the use of tuberculous meat."

Seeing in faulty environmental conditions and in the spitting habit the principal factors in the spread of tuberculosis in Hong Kong, the author recommends, as the first and greatest need in prevention,

a campaign of education as to hygiene, through the Chinese doctors who come into close touch with the people on the one hand, and by means, perhaps, of the Y.M.C.A. and Y.W.C.A. on the other. Finally, he advocates the initiation of a "Tuberculosis Dispensary." As he wisely expresses it, "Enlightment is not only better than repression, but is really the only method likely to be crowned with any success."

In the second portion of the Report are considered the portals of entry and mode of spread of tuberculosis in the light of the work carried out by the author, and, in the third portion, he discusses the morbid anatomy as met with in cases among children. As these two portions of the Report are complementary to each other, they will be discussed together.

"Of the whole 300 cases, there were 209 in which the primary portal of entry appeared to be the respiratory tract, *i.e.*, 69·66 per cent., and in 7 others . . . there was considerable evidence in support of the same portal, bringing the total to 216, or 72 per cent.

"Only 32 have been definitely determined as being of alimentary origin, *i.e.*, 10·66 per cent.; five more, discussed under 'Uncertain primary portal' were very likely alimentary; this would bring the total to 37 or 12·33 per cent.

"In four other cases there was a possibility of almost simultaneous entrance by way of the respiratory and alimentary tracts."

In the 43 remaining cases the primary portal of entry is regarded by the author as uncertain. These uncertain cases are discussed in individual detail and the reader is thus enabled to form his own opinion as to the probable mode of entry of the bacillus in each patient. So far as we could judge from a careful perusal of these excellent and careful records, the majority were, in all probability, cases of invasion by the respiratory route, but we concur with the author in his decision to base his conclusions, as far as possible, only on undoubted cases, and we agree as to the wisdom of keeping these less definite cases apart.

Part III really consists in a comparison of the findings of the author with those of Canti. On the whole the agreement is close, but there are certain points in which differences arise. So important is this portion of the work that it requires notice in some detail. Following the method of the author of taking Canti's conclusions in order and comparing his own findings in each case, we will attempt to summarize his work on the same lines.

1. "The almost constant finding of a lung focus when tuberculous mediastinal glands are present and the close relation of these glands to the lung focus" (Canti).

Dr. Scott finds this statement to be true of the majority of the cases examined by him, but he notes 29 exceptions. Of these, 12 are excluded, 11 having strong evidence of primary alimentary infection, and one "having a tuberculous abscess of the sixth cervical vertebra discharging into the right pleural cavity which might explain the involvement of the mediastinal glands." There remain 17 cases in which the mediastinal glands showed definite tuberculous lesions without any discoverable lung focus to explain the adenitis. Particulars of all these cases are given in detail and they are worthy of careful perusal and close consideration. As to the "close relationship of these glands to the lung focus," the author found this to apply in most cases, but there were four in which the glands involved did not correspond with the focus in the lung.

2. "The frequent singleness of the lung focus" (Canti).

In 16 cases described by CANTI eight, or 50 per cent., had a single focus. GHON recorded 72·35 per cent. Dr. Scott found, in 225 children under 10 years of age, 137 with lung foci. Of these 95, or 69·34 per cent., had a single focus. There were 42 cases, or 30·66 per cent., with more than one focus. Of the latter, 32 had two, 5 had three, and 5 had several foci in the lungs. The distribution as to lobes in these 137 cases was as follows :—

Right upper lobe	42
Right lower lobe	41
Left upper lobe	32
Left lower lobe	26
Right middle lobe..	22

3. " The constant finding of tuberculous mediastinal glands where a lung focus was present " (CANTI).

The author remarks : " In adult cases, this has certainly not been my experience here, but it appears to hold good with nearly all cases in children." He quotes nine cases, however, of children in which lung lesions were present without coexistent disease of the mediastinal glands.

4. " The almost constant absence of a lung focus when the portal of entry appeared to be elsewhere " (CANTI).

Excluding cases in which a dual route of infection appeared probable, Dr. Scott says " There still remain a few in which a lung focus was found although the primary portal of entry was probably not *via* the respiratory tract." He quotes these four cases in detail, but we are obliged to admit that, so far as the notes go, they appear to us to point to infection through the respiratory rather than through the alimentary tract. This must, however, remain a question of opinion until fuller knowledge of the responses of the body to infection is available.

5. " The almost constant absence of evidence that the portal of entry might be elsewhere when a lung focus was present " (CANTI).

On this point the experience of Dr. Scott is distinctly at variance with that of Dr. CANTI. As he expresses it :

" Such a proposition would seem to put forward the claim that, given a primary lung focus due to respiratory route infection, there is little, if any, likelihood of the intestine becoming affected unless secondarily to the pulmonary focus. This cannot be ascribed to an increased resistance or immunity, owing to the presence of the lung tuberculosis, because it is a well-established fact that intestinal tuberculosis can arise from the swallowing of infected sputum."

He gives several instances which he thinks do not fit in with CANTI's proposition.

For our own part we hold that sensitization of the organism by initial entry of the tubercle bacillus is essential before local reaction can follow fresh contact with the germ. The experiments of CALMETTE, GUERIN, and BRETON, quoted by the author, prove that the tubercle bacillus can penetrate the membranes of non-resistant animals (guinea-pigs) without setting up lesions, coming to arrest in the lymphatic glands beyond. It is *after* such preliminary penetration that inflammatory reaction to the advent of further bacterial invasions leads to focal changes in the lung or ulcerative changes in the intestine. It is thus that we should explain the instances cited by the author of involvement of mediastinal glands without lung lesions in occasional cases of massive infection ; and it is for the same reason that we regard

ulcerative lesions of the bowel in tuberculosis as a sign of *previous* infection, not as a sign of *primary* infection of the alimentary tract.

It is not, however, our desire to attempt to criticize the conclusions of the author but rather to pay a high tribute of praise to one of the most thorough and comprehensive investigations that we have yet met with on a very important sphere of research in tuberculosis.

The fourth portion of his paper consists of careful details of each case examined and is a model of what such records should be. His comparison of post-mortem findings in Chinese children with the findings of Dr. Canti, in London, and those of Albrecht and Ghon, in Germany, is particularly useful and points to a line of research that might be profitably followed in other colonies and protectorates of the British Empire.

S. Lyle Cummins.

Scott (Henry Harold). **The Prevalence and Character of Tuberculosis in Hongkong. Parts 1 & 2.**—*Ann. Trop. Med. & Parasit.* 1921. Sep. 30. Vol. 15. No. 3. pp. 213–243. With 1 text fig.

Since the receipt of this Report in manuscript from the Colonial Office, the greater part of the work has appeared in the *Annals of Tropical Medicine and Parasitology*. As the summary given above covers the published articles, no further review is necessary, but those interested in tropical tuberculosis are strongly recommended to peruse the papers now available for study; they constitute an important contribution to this subject, as well as to our knowledge of tuberculosis in general.

S. L. C.

Mitchell (J. Alexander). **The Problem of Tuberculosis in South Africa. (Address given at the Congress of the Municipal Association of the Cape Province, Cape Town, March 8, 1921.)**—*S. African Med. Rec.* 1921. June 25. Vol. 19. No. 12. pp. 226–230.

Dr. Mitchell shows how tuberculosis, rare or unknown in the early days of the Colony, has become more and more frequent as communications have improved, towns developed, industries become established, and native populations, formerly isolated, been recruited for the mines and for other work involving adaptation to a new mode of life. Tuberculosis has been notifiable in the Cape Province since 1907, and throughout the Union since the passing of the Public Health Act in 1920. The statistics, however, are far from reliable, the percentage of unnotified cases being very high. "The great majority of the sufferers are young adults, who are thus disabled, and in many cases killed, at the age when they are of the greatest economic value to the State." The author has great hopes for the future of anti-tuberculosis measures so far as the protection of Europeans is concerned, but "as regards the Coloured and Native, the outlook is much less hopeful, mainly owing to their susceptibility to the disease and the difficulties of enforcing precautions against its spread." The author lays much stress on alcohol as a potent factor in the prevalence and spread of tuberculosis amongst the coloured folk. He is not alone in this opinion. It is held by nearly all writers on the tuberculosis of primitive peoples. And yet we are unaware of any really critical investigation that gives the theory support. The susceptibility of these African tribes is paralleled by the susceptibility of cattle from tubercle-free localities when brought into contact with infected herds;

and yet, as CALMETTE so wittily pointed out at the recent International Conference, "It is difficult to believe that the prevalence is due to alcohol amongst these poor beasts."

S. L. C.

YOUNG (J. F.); GIRDWOOD (A. I.); & others. [**Tuberculosis: Discussion on Tuberculosis as found on Examination of British South African Natives and of East Coast Recruits.**]—*Proc. Transvaal Mine Med. Officers' Assoc.* 1921. Aug. Vol. 1. No. 5. pp. 2–11.

The discussion here summarized was opened by Dr. J. F. Young, Medical Officer, Native Recruiting Corporation, Driehoek, who spoke on the examination of B.S.A. natives. He began by describing the methods of medical examination in force at the branch offices of the Native Recruiting Corporation in Cape Colony and Pondoland. A doctor is attached to these offices to examine the mine recruits. At the smaller branch offices the natives are merely inspected, but at the larger ones "they are usually auscultated." For the year ended on 31st December, 1920, 30,554 natives were examined, and 807, or 2·64 per cent., were rejected for "defective lungs," and, in addition, 56 for tubercular glands. Dr. Young estimates that 75 per cent. of the "defective lungs" were tuberculous cases. In another group of 36,243 natives examined, 738, or 2 per cent., had tuberculosis. "During 1919 at Sterkstroom all natives rejected for defective lungs were sent back home." For the year ended May, 1921, at Germiston, 62,942 natives were examined. Of these, 1,075, or 1·7 per cent., were rejected for "defective lungs," but the number of these suffering from tuberculosis was not differentiated. In the course of the paper the remark is made that British Basutos are far less afflicted with "defective lungs" than the Colony natives.

Dr. Girdwood, Chief Medical Officer, Witwatersrand Native Labour Association, now contributed a paper on the examination of East Coast Recruits, and explained that the system of examination of these recruits was somewhat superficial as compared with that of the British South Africans. His paper, however, constitutes a very valuable contribution and shows a real familiarity with his subject. The East Coast natives are recruited from the Province of Mozambique, south of lat. 22°. Throughout the year 1920 a total of 60,164 East Coast natives were examined. Of these, a total of 1·86 per cent. were rejected for all causes. Of 462 rejected by Dr. Girdwood, only 83 were suffering from tuberculosis. Post-mortems on 39 new recruits who died before working only brought to light two cases of tuberculosis. "Out of the 174,402 natives employed on the gold mines during 1920, 28 were compensated for tuberculosis or silicosis during the first two months of their contract. The number compensated steadily increases, to reach a maximum during the fifth and sixth month of their service." Dr. Girdwood is of opinion that pulmonary tuberculosis is not common amongst East Coast natives, and he cites Dr. TURNER, who found, in 1905, that only 14 cases could be detected after an extensive tour of the district, 10 of which proved to be natives who had returned from the mines. These East Coast men are much more susceptible than the British South Africans, and, once at the mines, evince a terrible liability to the disease, explicable, according to Dr. Girdwood, on one of the following hypotheses: (1) that the disease may be a new one to them so that they have not yet acquired an hereditary immunity to it; or (2) that they have enlarged spleens and malaria, and thus a lowered

resistance ; or, again (3) that the length of contract, one year, may have something to do with it.

Dr. ALLEN now spoke, and agreed with Dr. Girdwood that tuberculosis is extremely rare among new recruits coming up for the first time. He was of opinion that the infection among the natives is acquired on the mines. "The compound, congregated as the boys are in it, is the focus of infection." Dr. BOIGELOT, Medical Inspector of Native Labour for the Congo, now spoke, and made an interesting communication as to the great rarity of tuberculosis amongst the natives in his area, except in the immediate vicinity of infected persons from outside. He laid stress on the value, as an index of good health in natives, of the shine and brilliancy of the hair, the colour and glow of the skin and its elasticity when pinched up. Dr. BROOKE now spoke, and explained that, in the native territories with which he was acquainted, tuberculosis was on the increase, largely due to the taste for education developed by the natives and the crowded conditions in their schools and association with infected school teachers. He raised the question of the difficulty of examining more than 40 or 50 chest cases in a morning. He also spoke on the difficulty of early diagnosis, and mentioned the frequency with which indefinite crepitations are found along the margins of the lung in the native.

Several other interesting contributions were made to the discussion, notably by Dr. ORENSTEIN and Dr. FREW. Referring to Dr. Brooke's remark, Dr. Young said that he had often to examine as many as 560 or more natives in the morning.

The impression gained on reading this discussion is that the examinations of these recruits are of a perfunctory nature and that the work is far in excess of what can be properly carried out by the staff available. It is clear that the whole system by which native labour is recruited, exploited and returned, often infected, to its original surroundings when no longer fit for work is eminently calculated to spread the disease far and wide in the crowded huts of insanitary native villages, where the disease was, until recent times, unknown. That this is actually happening may be gathered from the remarks of Dr. Brooke, and the conditions that assure a rapid spread are shown, by Dr. Girdwood, to exist in a typical form in the districts where the East Coast recruits are obtained. These conditions are an absence of any considerable prevalence of tuberculosis in a given area, a race with a high susceptibility to the disease, and the return of infected cases from the mines. One would imagine that the mining companies themselves ought to be alive to the unscientific nature of such methods, which can only end in destroying the quality and diminishing the quantity of available labour.

S. L. C.

ROBERTSON (D. G.). **Inquiry into the Prevalence of Tuberculosis at Bendigo.**—*Commonwealth of Australia Quarantine Service Publications.* 1920. Mar.–Aug. No. 19. 70 pp. With 12 text figs.

Owing to the loss of life during the war, the Commonwealth Government, in 1916, appointed a Committee to enquire into, and advise as to the best means of preventing, death and invalidity in the Commonwealth itself. This Committee, in regard to tuberculosis, made the following suggestion :—"That the Commonwealth Government co-operate with the State Government concerned in a vigorous campaign against tuberculosis in a selected locality in which it is more than

ordinarily prevalent." The mining centre of Bendigo was selected for this experiment because it offers the chance of attacking not only the general problem of tuberculosis but also the special problem of tuberculosis in mines.

The article under review deals with the preliminary enquiry as to tuberculosis in Bendigo district and includes recommendations as to how the problem should be dealt with. The Report is of the highest interest and value, not specially as a contribution to "tropical tuberculosis," as the population concerned is of European extraction and lives under the conditions of industrial life common to workers elsewhere, except for the fact that there is a terribly high incidence of silicosis due to mining in quartz, but as a contribution to the general problem of industrial hygiene. The paper is so interesting that it merits a very complete notice, but, in view of its slight bearing upon tropical medicine, its main points only will be summarized at the present time. Under the heading of "Statistical Data" an interesting account is given of tuberculosis in Bendigo from 1905 onwards. Charts A, B, and C illustrate graphically the annual notifications of pulmonary tuberculosis in the area from 1905 to 1919, the two latter comparing the notifications of males with those of females. These charts bring out the interesting fact that, while up to the age of 25 to 30 the incidence amongst females slightly exceeds that of males of the same ages, the male curve continues to rise to an apex between 40 and 44, after which it falls rapidly, the female curve dropping steeply after 30. It would seem that very few females are affected late in life, while some factor operative in males leads to a preponderating incidence in middle age. This factor is silicosis, which tends in Bendigo, as elsewhere, to greatly increase the phthisis death-rate. The paper contains one of the best clinical accounts of silicosis that we have seen and the further note on "tuberculosis complicating silicosis" is likewise very good. The author takes the generally accepted view that the tuberculous condition is a superinfection of the lung already injured by silicosis. There must be ample opportunity for infection in the Bendigo mines as no less than 18 or 3·15 per cent. of 570 working miners were found, on examination, to be suffering from tuberculosis, while 41·25 per cent. of 160 ex-miners had the disease. The close association between the degree of silicosis and the incidence of tuberculosis is well brought out by the records in groups with and without machine drill work. Those who work with the drill are exposed to more concentrated dust, get more silicosis, and have a higher incidence of tuberculosis than the others.

There is an interesting note on chest expansion which gives results as follows:—In 345 healthy men, average expansion, 3·2 in.; in 150 "early silicosis" cases, 2·4 in.; in 54 "intermediate silicosis cases," 1·9 in.; in 3 "advanced" silicosis cases, 1·1 in., and in 18 tuberculosis cases, 2·2 in.

A valuable record of Von Pirquet tests in miners and non-miners is also given, of which the main results are as follows:—

	Total.	Von Pirquet —
1. Persons classified as tuberculous	60	55 or 91·7%
2. Miners and ex-miners (Group 1 excluded)	37	21 or 56·7%
3. Contacts with tuberculous persons	226	135 or 60·0%
4. No history of exposure to TB	69	16 or 23·2%

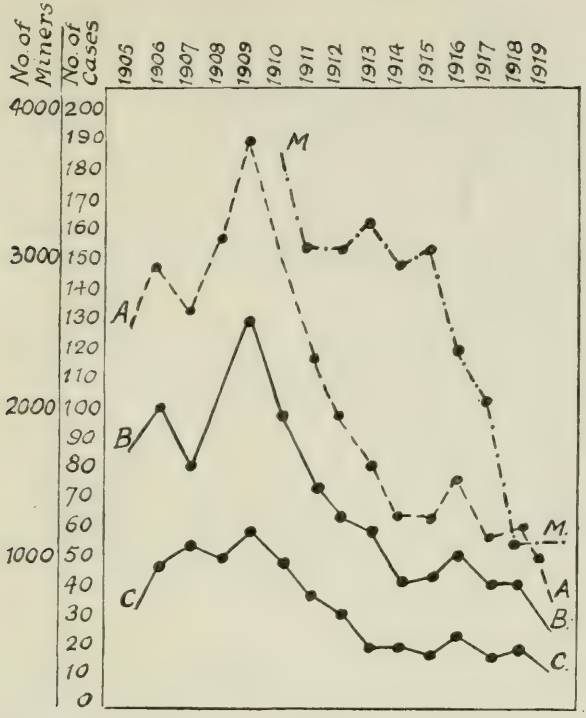


Chart A.—Annual Notification of Pulmonary Tuberculosis. Bendigo District, 1905–1919.

AA. Total Notifications. CC. Female Notifications.
BB. Male ,, MM. Number of Miners employed.

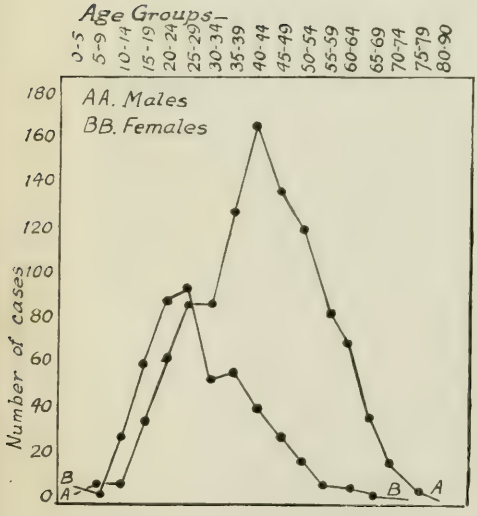


Chart B.—Notification of Pulmonary Tuberculosis, Bendigo District, at Various Ages.

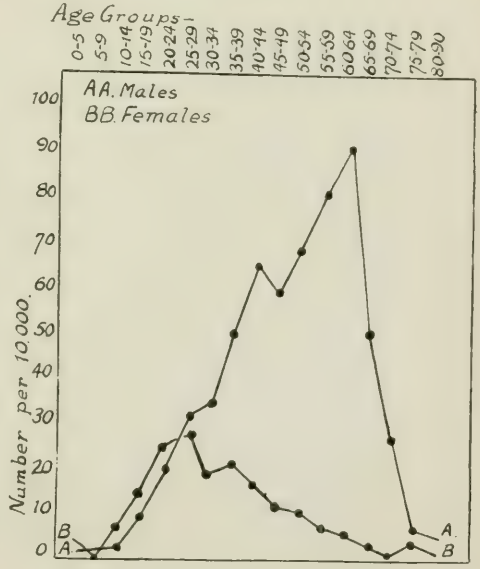


Chart C.—Average Notification Rates, 1905–1919, of Pulmonary Tuberculosis per 10,000 living at each Age Period in the Bendigo District in 1911.

The recommendations include the provision of a Sanatorium and a Clinic, a Medical Officer and a Nurse, the services of a half-time Radiographer from Bendigo Hospital, better financial help to families where the breadwinner is incapacitated by tuberculosis and the immediate introduction of a Miner's Compensation Act to meet the incapacity caused by silicosis, a purely occupational disease.

S. L. C.

COLOMBANI with VUILLET. **Le problème de la tuberculose au Maroc.** —*Bull. Office Internat. d'Hyg. Publique.* 1921. Apr. Vol. 13. No. 4. pp. 360–366.

"Il y a peu d'années encore, la population marocaine semblait particulièrement favorisée quant a la fréquence de la tuberculose pulmonaire." It is the old story: a country free or almost so from this disease until the arrival of the European civilization that brings with it the healthy carriers of infection, the advanced cases in search of a climatic cure, the industrial developments that require native labour to exploit them, and the invitation to participate in a Western mode of life that seems so attractive to the African. And the disease soon gains a footing and is not slow to exploit the virgin soil of these tribesmen who fall an easy prey to its ravages. The rapidity of its spread in Morocco may be gathered from Table I, which is here reproduced.

Pulmonary Phthisis at the Tangier Hospital. 1900–1919.

Year.	*Number of Admissions for Pulmonary Phthisis.*	*Number of Deaths from Pulmonary Phthisis.*
1900–1903	15	2
1904–1907	28	4
1908–1911	88	22
1912–1915	96	22
1916–1919	121	33

The authors find an explanation of this rapid spread in the profound changes that have taken place in the economic and social life of a population until recently outside the movements of our modern civilization. They quote the immense strides made by sanitary science in Morocco. "So far as concerns malaria, syphilis, dysentery, enteric fever, plague or typhus, to quote only a few examples, the results obtained have been as satisfactory as possible." . . . . "Pourquoi la tuberculose fait-elle exception dans cette amélioration générale?" The authors answer their own question by quoting the words of LANDOUZY: "Social evolution goes ahead so fast that hygiene, soon outdistanced, gets left behind." Yes, this is all very well and the quotation is attractive; but why is it only that the hygiene that might deliver man from tuberculosis gets left in the lurch while the hygiene appropriate to prevent typhoid, typhus, plague, malaria, etc., keeps pace with other forms of human progress? The truth is that, in all these diseases, the hygienist has been taught by the laboratory worker some special method of prevention that is efficacious while, in the case of tuberculosis, the special method has not yet been found and we flounder in the morass of general hygiene which is unequal to the stamping out of this difficult foe.

The spread of this disease is very little affected by our preventive methods either in Africa or in Europe. The mortality from the

chronic form of phthisis common in our cities does vary with the general death-rate and falls where the general standard of hygiene is high, but this is because *infected persons* die if exposed to a bad environment. The "young adult" type of mortality, common in Ireland, Wales, the Shetlands, and other relatively isolated places in the West, and comparable to the type of death-rate in Morocco, is not found to vary with the general death-rate or with the state of efficiency or otherwise of the sanitary organization of the community, but requires to be explained in terms of liability to infection, not liability to relapse. We fear, too, that the inevitable appeal to alcohol as a valid explanation will hardly serve to account for all the facts. Rather do we turn to such investigators as CALMETTE, BUSHNELL and ZIEMANN for the work that they have done on the influence of acquired immunity as the fundamental factor behind these phenomena. The authors have written a valuable and interesting paper and we heartily endorse their recommendation for an energetic campaign against tuberculosis in Morocco.

S. L. C.

TRABAUD. **Mémoires sur la tuberculose dans l'armée et dans les troupes indigènes et sur la prophylaxie de cette affection.**— *Arch. Méd. et Pharm. Milit.* 1921. Vol. 74. No. 4. pp. 432–436.

This paper is an analysis, by PERDRIZET, of an original report by Trabaud, submitted to the Technical Section of the Army Medical Service. It is to be hoped that the original Report may, in the future, be published in full, as the summary gives promise of a highly interesting and valuable paper. Trabaud deals with what is evidently a rich collection of clinical and post-mortem records under three heads: tuberculosis in the Senegalese, tuberculosis in Arabs, and, finally, a comparison of tuberculosis of the white races with that of Primitive races. In his description of the disease, as it affects the natives of Senegal, his findings correspond closely to those of BORREL (this *Bulletin*, Vol. 16, p. 190), but he considers that the statement of the latter that the presence of a palpable enlargement of a subclavicular gland is a sure sign of pulmonary tuberculosis is too exclusive, since this sign often indicates abdominal tuberculosis, while, on the other hand, axillary adenitis is often a sign of pulmonary or pleural infection. Where a subclavicular gland is found enlarged as a result of abdominal tuberculosis, Trabaud takes the view, with KUTTNER, that the tuberculous embolus has followed the collateral path indicated by the latter observer, passing by an efferent branch of the internal mammary chain of lymphatics that leads into the subclavicular glands of the infero-internal group; and he records two cases in support of this opinion. These observations lead to the conclusion that tuberculous infection in the Negro is not confined to the pulmonary route, but often starts by ingestion of the bacilli and invasion through the alimentary tract. The outstanding feature of tuberculous infection in the Negro is its predilection for the lymphatic system, the glands tending to react by a rapid and voluminous caseation, as in the younger age groups in Europeans. With this goes a tendency to extensive caseous pneumonia, with the final formation of ragged soft-walled diffluent cavities, contrasting sharply with the indurated and regular walls of the cavities commonly found in European adults. A clinical finding noted by the author, and not quite in line with the usual post-mortem findings in blacks, is that definite signs of disease can be made out at the apices in 85 per cent. of cases. He lays stress on the loss of satiny lustre in the black skin and a general flabbiness

of the muscles, which are valuable and early indications that the patient has contracted tuberculous disease.

In contrast with the natives of Senegal, the Arabs show a higher resistance to the tubercle bacillus, but are less protected than Europeans, the usual type of lung lesion seen at post-mortem being scattered foci of caseation corresponding to broncho-pneumonic patches of different sizes, but not, as a rule, larger than an orange. The glands of the hilus, too, are often enlarged and caseous, but less so than in the Negro. The Arab, then, occupies a place between the Negro and the European, and probably owes what extra resistance he possesses to contact with the latter. In comparing the manifestations of the disease in primitive races with its course in Europeans, Trabaud finds that the picture presented by the disease in these peoples bears a close similarity to that noted in European infants, and in this view he voices an opinion already expressed by Borrel. The paper closes with an excellent scheme for prevention of spread of infection in "Native" military formations, which deserves attention by those serving with troops drawn from isolated communities in the tropics or elsewhere.

S. L. C.

British Guiana. **Fourteenth Annual Report of the British Guiana Society for the Prevention and Treatment of Tuberculosis.** [Minett (E. P.), Hon. Secretary.] 1920.—18 pp.

This report, the fourteenth published by the Society, is chiefly concerned with the administrative measures and the arrangements for finance and staff, which are always an important feature in such organizations. There must, however, be a great deal of interesting information to be had in a community where the population includes several racial strains, and it is to be hoped that questions relating to the incidence and mortality in Africans, East Indians, Portuguese, British, etc., may receive attention in future reports. The patients seen at the dispensaries at Georgetown, New Amsterdam and Schoonord are dealt with under age groups, races, occupations, etc., but these figures give no light on the relative severity of the disease in these groups, as only the people attending the dispensaries are dealt with, and the population from which these patients are drawn may be large or small, rich or poor, susceptible or resistant, for all the statistics tell us. The retrospect of the tuberculosis death-rate per 1,000 persons living from 1876 to 1920 is of decided interest, and is encouraging also, as the mortality for British Guiana has fallen from 2·36 from 1876–1881 to 1·34 in 1920. The death-rate in the towns—Georgetown and Port of Spain—has always been higher than that of the whole community. It was 5·9 in Georgetown in 1903 and only 2·9 in 1920; the latter figure, though high, representing a very marked drop in such a short period. It is clear that British Guiana is taking the tuberculosis problem seriously, and that her efforts are meeting with a satisfactory measure of success.

S. L. C.

Fontes (A.). **Prophylaxia da tuberculose.**—*Brazil Medico.* 1921. July 2. Year 35. Vol. 1. No. 27. pp. 344–350. With 4 text figs. July 23. Year 35. Vol. 2. No. 2, pp. 13–19. And Sept. 3. Year 35. Vol. 2. No. 8. pp. 98–102.

This highly valuable paper is written from the point of view of one who has taken a comprehensive survey of the whole field of

the epidemiology of tuberculosis, and who brings to the study of the question a wide knowledge of the literature of this disease, as well as a close practical familiarity with its manifestations in man and animals throughout the world. He has no illusions as to the efficacy of the conventional methods of the general sanitarian in the war against tuberculosis. The fall in the mortality from this disease, that has been such a marked feature of its epidemiological history from 1800 onwards, he explains on the grounds of acquired immunity, and he takes pains to point out that the diminution was already well advanced several years before there was any real system of public health administration in being. He deals severely with the suggestion that the isolation of infected individuals can ever be applied with sufficient thoroughness to be effective, and the arguments that he uses are somewhat as follows :—

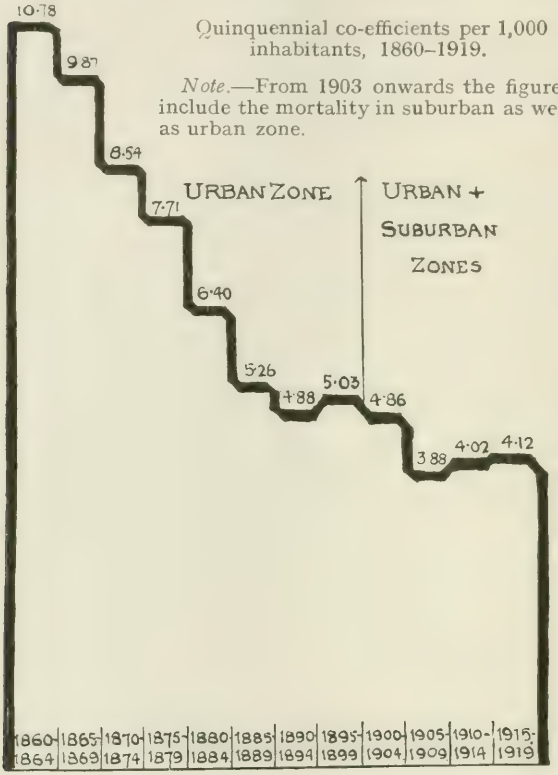
" Von Pirquet " tests and P.M. statistics indicate that nine-tenths of the population of large towns are infected with tubercle bacilli. Of these, it must be supposed that at least 50 per cent. disseminate bacilli in the sputum or otherwise. Taking Rio de Janeiro as an example, with a population of 1,157,873 inhabitants, at least 1,042,085 persons are likely to be infected, from amongst whom about half, or 521,000, are in all probability excretors of the germ from time to time. Is it reasonable, he asks, to advocate the isolation of over 500,000 people in order to go some way to lessening the 4,500 deaths caused annually by the disease in the town of Rio.

Without quite accepting the validity of the induction as regards the actual numbers assumed to be infected and infective, there can be no doubt that the general line of the argument is sound. The carefully supported statements of CALMETTE at the recent International Conference on Tuberculosis in London have called attention to the importance of the excretion of tubercle bacilli in the stools of a large number of apparently healthy persons, and it is certain that the numbers of " open cases " do not by any means represent the whole of the active disseminators of tuberculous infection. In his desire to expose the futility of some of the more widely advocated measures of prophylactic administration, the author sometimes goes too far. We think that his criticism of the regulations making notification of cases compulsory is unsound. This measure lies at the root of sound preventive measures, as it gives an approximate measurement of the problem and affords facilities for the examination of " contacts " and the removal of young children from a dangerous environment. Reproductions of two Anti-tuberculosis Decrees, one dating back to 1733 and the other to 1767, serve to illustrate the fact that we had " general sanitarians " as long as 200 years ago, and that what they advocate now they advocated then. Dr. Fontes draws a sharp line of division between tuberculous infection and tuberculous disease. Nine-tenths of the population, he says, of civilized communities are infected. Only a relatively small number show signs of the disease. He finds insensibility to the virus a sign of relative immunity to the disease, and recapitulates the oft-quoted instances of the ravages of tuberculosis when introduced for the first time into communities free from previous infection and representing " virgin soil." A chart illustrating the steady fall, down to its present (still high) level, of mortality from tuberculosis in Rio de Janeiro is of considerable interest and is here reproduced.

In the second portion of the paper the tuberculosis of childhood and adolescence receives attention, and the different types of

clinical manifestation of the disease in the varying age-groups, as well as the mortality in relation to age, are dealt with in a very thorough manner. The increase of the mortality from tuberculosis associated with advancing years is confirmed by extracts from the statistical studies of many authors. The question of heredity and hereditary predisposition is discussed, and the view is taken that the increased mortality amongst the children of infected parents is associated with infection at a susceptible age rather than with an inherited disposition, but the possibility of a transmission of resistance to infection as an inherited character is looked on with more favour.

Mortality from Tuberculosis in Rio de Janeiro.



Discussing the infection of adults, the author points out that the breaking down of resistance and the appearance of active disease is not always due to reinfection, but often attributable to alcohol, self-indulgence, acute infectious diseases, and so on. Much stress is laid upon the results that may be expected to follow education of the masses in personal hygiene, and the wise proverb is quoted: "Onde nao entra o sol, entra o medico; e onde nao penetra o ar, penetra a morte." (When the sun is shut out, the doctor comes in; if the air is kept out, death enters.)

In the number of 3rd September Dr. Fontes concludes his observations upon tuberculosis and deals with the question of

prevention. Having in view the extremely wide diffusion of the disease, tuberculous infection may, in the opinion of the author, be regarded as practically universal amongst those who pass their lives in urban communities, and for this reason resistance acquired through involuntary immunization amounts to a preponderating factor in the defence against the development of phthisis.

Dr. Fontes draws from this conception the natural inference that preventive measures should be directed for the most part to the protection of the still susceptible groups of children and adolescents. He advocates a much larger measure of assistance for the mother during the period of gestation and a more efficient system of puericulture. His opinion of existing methods of education in the elementary schools of his country is not high, and he is in favour of radical changes directed to the production of healthier minds in healthier bodies. In order to deal scientifically with the tuberculosis problem, he suggests a comprehensive scheme for the classification of school children on a basis of tuberculin tests followed by careful clinical investigation, illustrating his idea by means of a formidable diagram. While we agree with his scheme in a general way, we fear that it is too theoretical to be of much value in actual school hygiene. It is undoubtedly true that all school children should be submitted to the von Pirquet reaction at intervals, and that the information so gained should be applied in the direction of closer observation and the provision of more open air and shorter hours of work for those that show an early "positive" response; but there is a danger that this test may lead to groundless alarm and undue interference with education unless the supervision is in very wise and capable hands, and we are not optimistic as to the prospects of its judicious interpretation by the average schoolmaster or schoolmistress.

The paper finishes with an ambitious suggestion for the creation of a "Supreme Council of Prophylaxis and Assistance," and here, too, we feel that the author is rather inclined to see visions and dream dreams. But nothing great is ever accomplished without imagination, and perhaps the future may see the initiation of public bodies with programmes of ideal perfection and constituted of human beings worthy of the task. In the meantime, Dr. Fontes is to be congratulated on a very able and thoughtful paper. With his general arguments and recommendations all students of tuberculosis must agree. "Let us make war on the germ," he says, "but, above all, let us ensure sound men for the future of our race by preserving and developing the infants of to-day." These are words of wisdom, and we only wish that they were printed in letters of fire over the offices of every sanitary authority.

S. L. C.

DAVIES (Caleb). **The Value of Sodium Morrhuate and Sodium Linate in Tuberculosis and Leprosy.**—*Indian Med. Gaz.* 1921. Aug. Vol. 56. No. 8. p. 283.

The author has used sodium morrhuate and sodium linate in the treatment of both tuberculosis and leprosy, and believes that he has obtained satisfactory results. In the short paper now under consideration he speaks chiefly of his results in tuberculosis, which appear to have been favourable, and he notes the fact that he observed what he judged to be focal reactions in some of his cases after the morrhuate.

With the linate, the result seems to vary a great deal with different batches of this preparation, some samples proving quite non-irritant in the hands of the author, though Sir Leonard ROGERS reports that he found that the linate used by him was irritating when given subcutaneously. The same would appear to apply to the morrhuate.

S. L. C.

LINDENBERG (Ad.) & RANGEL PESTANA (Bruno). **Chemotherapeutische Versuche mit Fetten an Kulturen säurefester Bacillen.**—[Chemotherapeutic Experiments with Fats on Cultures of Acid-fast Bacilli.]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1 Teil Orig. 1921. Vol. 32. No. 1. pp. 66–86.

This highly interesting and important paper commences with a short account of previous work on the use of chaulmoogra oil and allied preparations which is, in one respect, curiously incomplete in that it omits any mention of the researches of Sir Leonard ROGERS and his co-workers in India. It is remarkable, too, that although sent for publication at the end of November, 1920, this paper fails to notice the work of WALKER and SWEENEY on a very similar aspect of the leprosy problem published in the *Journal of Infectious Diseases* in the preceding March. [See this *Bulletin*, Vol. 17, p. 277.] It would seem that the limits set to the study of current literature by the racial animosities of the war have not yet been entirely got rid of. The authors, wishing to obtain a preparation suitable for intravenous injection, proceeded as follows :—To a 2·5 per cent. solution of soap of chaulmoogra oil was added an equal volume of the original oil itself, the mixture being made in small volumes at a time, with careful shaking during the process. The whole was now diluted by the addition of an equal volume of distilled water, then passed through a Berkfeld filter and, finally, sterilized in the autoclave. A stable emulsion, seen under the microscope to consist of discrete elements, the smallest of which were of the size of pyogenic cocci, was thus obtained, and used with marked success in the treatment of leprosy. Notes of four striking cases are given. At this point an observation was made as to the content of the filtrate in free fat, and it was found that this had dropped from 7·7 per cent. before filtration to 4 per cent. after passing through the Berkfeld. It was evident that the greater part of the oil had been taken up by the filter. From the fact that the first filtrate still retained its therapeutic activity unaltered and that only the salts of the fatty acids remained constant after filtration, the authors drew the inference that the latter (actually the sodium salts of the fatty acids) represented the active part of the emulsion. This conclusion was confirmed by further experiment, and the authors now decided to attempt to work out the degree of activity by experiments *in vitro*.

To meet the difficulty presented by the fact that the leprosy bacillus has not yet been isolated, they undertook their experiments with other organisms of the acid-fast group, thinking, in view of the group action of salvarsan on spirochaetes and of antimony on trypanosomes, that the results with one acid-fast would probably represent those with the others. In the experiments now undertaken, a 1 per cent. solution of the sterilized fatty acid under examination was set up in a diminishing series of dilutions with glycerine broth, in tubes containing 10 cc., to each of which an equal volume of bacillary emulsion was added.

Suitable controls were carried out and the tubes examined at intervals to see whether growth had taken place. In this way it was proved that the fatty acid of *Taraktogenus Kurzii* prevented the growth of avian tubercle bacilli in a dilution of 1 in 100,000, while phenol, tricresol, and resorcin had practically no effect in comparison, Potassium aurocyanide, however, was shown, as previously demonstrated by KOCH, to be even more active. Further experiments showed that this action of the fatty acids was demonstrable for human tubercle bacilli, the butter bacillus, Duval's bacillus and the Timothy grass bacillus, but that these acids were without effect on such germs as *B. anthracis*, *B. subtilis*, *B. typhosus* and others. In this, the work of the authors confirms the observations of WALKER and SWEENEY.

They now proceeded to examine the activity of other fatty acids of the chaulmoogra series and found them all effective, but that of *Hydnocarpus wightiana* least of the group. An interesting point is that, in opposition to the findings of WALKER and SWEENEY, they obtained complete arrest of the growth of acid-fast bacilli with cod liver oil. This finding is in line with the clinical observations of Sir Leonard ROGERS and, if confirmed, may prove of importance. Oil of bacon fat, cacao, oil of cocoa nut and pea oil were all found to be inert, while oil of cotton seed, linseed, poppy and, to a less extent, sesame were found active, as was also *Johanesia princeps*. The authors now arrived at the fact, already noted by others, that it was the unsaturated fatty acids that were the active ones in preventing the growth of acid-fast bacilli, but, while associating this character with molecular structure, they make no mention of the observations of F. B. POWER, whose researches have thrown so much light upon this point. They publish an interesting note on the action of these preparations in tuberculosis and find that there is always a reaction as manifested by a rise of temperature and increased cough. They sound a note of caution in the use of these drugs in advanced cases. In its effects on acid-fast bacilli they find an explanation of the specific action of cod liver oil in tuberculosis.

This paper is a valuable contribution to a subject that is daily assuming more importance through its bearing upon the treatment of diseases caused by acid-fast bacilli.

S. L. C.

SHAW-MACKENZIE (J. A.). **On the Mechanism of Immunization, with Special Reference to Lipase.**—*Jl. Trop. Med. & Hyg.* 1921. June 15. Vol. 24. No. 12. pp. 161–164.

The author holds the view that an increase of lipase or fat-splitting ferment in the tissues is a factor in the defence of the body in cancer and certain bacterial diseases. He notes with interest the application of the unsaturated fatty acids of the chaulmoogra series and of sodium morrhuate to the treatment of leprosy and tuberculosis by Sir L. ROGERS and his co-workers because he thinks that the beneficial results may be due to the stimulation by these preparations of the lipolytic activity in the tissues and to the destructive action of the lipolytic ferment on the fats and lipoids of acid-fast bacilli. To show that the gynocardate and morrhuate really bring about an increase in lipolytic activity, the author carried out the following experiment, in which the lipolytic activity is estimated by the amount of decinormal potash required to neutralize the fatty acids set free by pancreatic lipase acting on olive oil emulsion. 1 cc. of glycerine

extract of pig's pancreas diluted with 2 cc. of water and 5 cc. of olive oil emulsion were used alone and with the addition of the substances to be tested. The mixture was incubated at 37° C. for about 18 hours and then titrated, phenolphthalein being used as an indicator. The results are thus set forth :—

	cc.		N 10 KOH required. cc.
Pancreatic extract alone on olive oil emulsion	—	..	24·7
Addition of sod. oleate to extract	1·0	..	37·9
	2·0	..	39·5
,, ,, morrhuate	1·0	..	37·4
	2·0	..	39·6
,, ,, gynocardate ..	1·0	..	35·2
	2·0	..	37·4

" It will be seen that all three salts exert marked accelerating action on the lipase of extract of pancreas." Dr. Shaw-Mackenzie has tried the treatment of active pulmonary tuberculosis by the intravenous injection of common animal soap in doses of 2 cc. of 2 or 3 per cent. solution, and he mentions the good results believed to have followed a similar kind of treatment by Dr. Dundas Irvine and Dr. C. W. Anderson. The paper is interesting and suggestive.

S. L. C.

LEPROSY.

LEGER (Marcel). **La lèpre dans les colonies françaises.**— *Ann. de Méd. et de Pharm. Colon.* Paris. 1920. Dec. Vol. 18. pp. 109–137. With 3 text figs.

This extremely interesting paper gives a comprehensive account of the distribution of leprosy throughout the colonial possessions of France. The material is arranged in a series of sections dealing successively with the French Colonies in Asia, Africa, America and Oceania. The disease is common in Tonkin, Cochin-China, Annam, Cambodia, Laos, and in the French territories of India. It is interesting to read that in Tonkin the natives, clearly recognizing that leprosy is transmissible from man to man, and regarding it as incurable, had for many years set aside special villages for the accommodation of cases. These villages included, in addition to huts for the inhabitants, rice-fields, fish-ponds and cultivated land on a scale intended to make the community self-supporting. The French administration, making full use of this system as a line along which anti-leprosy precautions might be further developed, has brought into existence "agricultural colonies" for all those cases whose means are insufficient to ensure adequate isolation and supervision in the domicile. The dominant idea in framing rules for the regulation of lepers has been to meet the native sentiments and to retain the goodwill of the inhabitants.

Turning to the African colonies, the author describes the distribution of the disease in French West Africa, including Mauretania, Haut Sénégal and Niger, Sénégal, Guinea, the Ivory Coast and Dahomey; French Equatorial Africa, with the mid-Congo and Chad provinces, as well as the recently acquired Cameroons; and French East Africa, including Madagascar, the Comoro Archipelago and Réunion. Leprosy is endemic in all these areas, though the incidence varies from an inconsiderable number, as in Mauretania, to 50 or more per 1,000 of the total population, as in parts of Senegal. In the village of Kissi, in Guinea, there are reported to be actually 48 cases amongst 150 inhabitants, or 320 per 1,000. With the exception of Madagascar, it may be said that none of these African dependencies possesses any satisfactory system of dealing with lepers. The African native has little liking for the restrictions of leper colonies, and there is no marked native prejudice against the association of the infected with the healthy. But in Madagascar there had been a clear recognition of the disease long before the French occupation, and under Ranavalona II an edict had been published directing that lepers should be isolated in special locations. General GALLIENI introduced extensive measures of isolation under conditions favourable to the patients, and this system has progressed until, in 1914, there were 4,021 interned patients out of an estimated total of 6,373 cases in the population of the island. In Réunion, where legislation, though of doubtful quality, has existed since 1852, the rules have invariably been evaded.

After a brief mention of the disease as it occurs in the French-American Colonies of the Antilles and French Guyana, the author passes on to a consideration of the colonies of Oceania, and here the subject takes on a new interest, since the history of leprosy in New Caledonia affords an opportunity of watching the behaviour of the disease in virgin soil. Introduced fifty-five years ago by a Chinaman, whose medical history has been well investigated by GRALL, leprosy spread

with such rapidity that, at one time, it threatened the inhabitants with extinction, even spreading to the Europeans, until 25 per 1,000 of these in the penal settlement and 14 per 1,000 in the free white population were affected. The efforts at the framing of anti-leprosy measures appear to have met with very little success. In spite of this, LEBOEUF, who carried out a careful survey of the distribution of the disease in 1911, 1912 and 1913, was able to draw the very encouraging conclusion that the disease was now undergoing a genuine diminution.* After an epidemic outbreak, following the introduction of the infection from elsewhere, the disease has tended to limit itself, while the infective nodular type has become less common and the anaesthetic form relatively more frequent. It is hard to avoid the conclusion that some degree of relative immunity has been acquired by the inhabitants, leading to the manifestations of the disease in its less virulent form of nervous leprosy. This idea is supported by the findings of VIALA in the Wallis Islands, where he " encountered very few cases of nodular type, but found that there existed in about half the population a form of attenuated leprosy, of which the chief signs were atrophy of the muscles of the hand and thickening of the ulnar nerve."

There is no more interesting feature in the history of leprosy than the fact that the disease has practically disappeared from Europe after a wide distribution in the Middle Ages, and that this disappearance had already taken place before modern hygiene had advanced sufficiently to be cited as the cause. The importance of this tendency to self-limitation in the spread of infective disease is very great. It is for this reason that, with reference to both leprosy and tuberculosis, the course taken by these diseases in New Caledonia merits the closest study by the epidemiologist.

S. Lyle Cummins.

DA MATTA (Alfredo). **Notas sobre a lepra no Amazonas.**—*Brazil Medico.* 1921. June 4. Year 35. Vol. 1. No. 22. pp. 287–291.

The author begins by expressing the truth that in discussing the transmission of leprosy two possible modes of infection have to be considered, the one problematical and in need of further investigation—that in which the intervention of an insect carrier is postulated, and the other well established and beyond doubt—the direct passage of the disease from person to person. A very complete summary, which we need not attempt to reproduce, is given of previous investigations as to insect vectors of Hansen's bacillus. The author, while admitting the weight of some of the evidence adduced, notably the researches of LEBOEUF with insects such as *Musca domestica*, as well as with fleas and bed-bugs, wisely preserves a critical attitude, remarking that if any of the more common insects were an effective transmitting agent, leprosy would spread far and wide in the valley of the Amazon.

Turning to direct contagion from man to man, Dr. Da Matta lays stress on the long time that may pass before exposure to infection finally leads to clinical manifestations, eight or ten years being often necessary. He points out, however, that an advanced case of leprosy is a very formidable focus of infection, the bacilli being disseminated in the nasal secretion, the saliva, the vaginal mucus, the discharges

*For summaries of Leboeuf's papers see this *Bulletin*, Vols. 1, 2, & 3.

from the open sores, the sweat, tears, faeces, etc. Methods of prophylaxis, then, must be based on the following schematic lines :—

1. Elimination of foci—isolation { domiciliary. / in leprosariums.
2. Restrictions on the immigration of lepers.

A brief historical account of the measures taken to prevent leprosy in Manaos follows, and the author gives some statistics collected from the records at the Lepers' Home at Umirisal. These figures deal with 131 cases, of which 103 were males and 28 females. The age distribution was as follows :—

Under 10 years	4
11–20 ,, ...	18
21–30 ,, ...	32
31–40 ,, ...	31
41–50 ,, ...	24
Over 50 ,, ...	22
	131

The clinical types were as under :—

Nodular	40	or 32 per cent.
Anaesthetic	48	,, 36·6 ,,
Mixed	20	,, 15·2 ,,
Trophic	12	,, 9·1 ,,
Doubtful	11	,, 8·3 ,,

The paper ends with a warning against an equivocal or perhaps erroneous statement often made as to the extreme prevalence of horrible and revolting cases of leprosy in the valley of the Amazon. As the author wisely points out, recent investigation has shown that many of the more terrible conditions of loss of naso-labial tissue and deep cutaneous lesions are due to *Leishmania americana* and to blastomycosis. Leprosy, though a very important item in the pathological picture of the country, is not so prevalent as is commonly supposed, being actually rare in parts of the interior.

S. L. C.

Alcazár (Izak). **A Study of Leprosy in the Holy Land.**—*Boston Med. & Surg. Jl.* 1921. May 19. Vol. 184. No. 20. pp. 518–521. With 4 text figs.

This paper gives a vivid account of the lepers in Palestine, and, while not attempting to deal closely with the medical problems of the disease, holds the interest of the reader through its sympathetic and humane attitude towards the social and historic aspects of leprosy. During the Turkish regime, lepers were allowed full freedom outside the walls of Jerusalem, and a regular leper community lived for many years in a cave on the slopes of the Mount of Olives near the village of Siloam. Their means of subsistance was by the collection of alms from tourists, and their operations were conducted on a definite plan under the direction of a chosen chief. They lived a communal life, marrying amongst themselves, making common arrangements for the division of their spoils and the purchase of food, and submitting all disputes to their leader, whose decision was final. As we read this brief account of a curious human agglomeration we cannot avoid being impressed

with the idea that this solution of the leper problem by the lepers themselves, may, after all, be the best and happiest arrangement. Better, perhaps, the "laisser faire" of the Turks than the leper islands and isolation establishments of less fatalistic peoples!

S. L. C.

Hijiya (S.) & Sakamoto (M.). **Das Leprosorium in Gotemba.**—*Japan. Ztschr. f. Dermat. u. Urol.* 1920. Sept. Vol. 20. No. 9. pp. 777–789. With 10 illustrations. [German summary p. 71.]

This is a brief account of the situation and organization of the Leprosorium of Gotemba, and gives no particulars as to the clinical side of the work in hand. The institute is placed at the foot of Fuji mountain at an elevation of about 300 metres above sea-level in an isolated and very healthy locality. First started by a French missionary named Testwid in 1889, it has dealt with 541 lepers (493 males and 138 females) to date; there are at present 70. The "mixed" type is predominant. The patients, other than those severely affected, are kept occupied with indoor and outdoor work of a suitable kind.

S. L. C.

Asano (Buichi). **Die Verteilung und die Statistik der Leprakranken in der Gegend von Aichi-distrikt.**—[Distribution and Statistics of Leprosy in and around Aichi Prefecture.]—*Hifuka oyobi Hinyôkika Zasshi (Japan. Ztschr. f. Dermat. u). Urol.* 1920. Sept. 20. Vol. 20. No. 9. pp. 789–806.

This is the result of the studies of the cases recorded in the Aichi Hospital of Nagoya, during the past ten years, November 1909–October 1919. The lepers dealt with are 814 in number, of which 79·4 per cent. are men and 20·5 per cent. are women. The majority of the patients belong to the "macular" type, being 424 in number (52 per cent.), while the "tubercular" and "mixed" types are the smallest groups, being each 44 in number (5·4 per cent.). The remainder belong to the "nervous" type.

As to the peculiar feature that the exposed parts of the body are those found mostly affected, and that the germ cannot grow on artificial culture media, the author suggests that the presence of light and the stagnation of venous blood may be necessary conditions for the growth of the germ, which latter seems to be aerobic in nature. The ages and occupations of the patients and the districts whence they came also receive consideration.

Hiroshi Ohshima.

Yamamoto (Tatsukichi). **Influenza Epidemic among Lepers (Double Infection).**]—*Tokyo Iji Shinshi* (Tokyo Med. News). 1920. Feb. 21. No. 2165. pp. 379–385.

The paper deals with the relation between leprosy and the influenza epidemic which prevailed in the autumn of 1918. Among 327 lepers under consideration there were 262 cases of influenza (80 per cent.) during the year beginning October 26th, 1918. Death occurred in 34 cases (less than 15 per cent.). Thus it seems that there exists no

difference in susceptibility to the epidemic between lepers and healthy people. The death-rate of less than 15 per cent. does not seem to be especially high. But as no reliable figures have yet been given as to the corresponding rates among healthy people, it would be premature to draw any definite conclusion.

Hiroshi Ohshima.

PERRET (J. M.). **Report on Two Cases of Leprosy.**—*New Orleans Med. & Surg. Jl.* 1921. May. Vol. 73. No. 11. pp. 473-475. With 1 text fig.

" Dyer in 1898 estimated the number of lepers in Louisiana as being between 300 and 500 "—(OSLER). The author concludes that there are probably more cases at the present day. He thinks that many are going about undiagnosed, and, as a matter of fact, actually found three cases in one day on visiting a dermatological clinic. In the present paper he describes two cases, both Louisianians, neither having ever left the United States. Good photographs are given. It is pointed out that the Wassermann reaction is often strongly positive in leprosy, and the author advises that leprosy should be suspected in cases with a positive Wassermann and yet with lesions that do not clear up with anti-syphilitic treatment.

S. L. C.

STEPHENS (J. W. W.) & ADLER (S.). **A Case of Suspected Leprosy.** —*Ann. Trop. Med. & Parasit.* 1921. July 16. Vol. 15. No. 2. pp. 173–177. With 2 text figs. and 1 plate.

This is a brief though very interesting account of a case of a patient who had, though born in England in 1904, resided in Capetown from 1905 to 1920. The symptoms and signs are well described and are those of maculo-anaesthetic leprosy. It is a matter of great interest that a very thorough search has so far completely failed to show the presence of acid-fast bacilli, though both scrapings and sections of tissue removed for the purpose have been investigated. In the excised portion of the enlarged and thickened ulnar nerve Prof. E. GLYNN reports the presence of three caseous areas, apparently corresponding to large nerve bundles and surrounded by fibrous tissue in process of formation, an appearance quite consistent with fibro-caseous leprosy.

We are inclined to think that the clinical and histological findings justify a positive diagnosis and appropriate treatment.

S. L. C.

CUSSEC (Jean). **Sur les caractères de l'anesthésie dans la lèpre.**— *Arch. Méd. et Pharm. Nav.* 1921. July–Aug. Vol. 111. No. 4. pp. 303–312.

This valuable contribution to the neurology of leprosy is a summary of observations made on 60 Senegalese soldiers embarked on the hospital ship " Flandre " for repatriation from Marseilles to Dakar, after having developed signs of leprosy during war service in France. Nine typical cases, illustrating an ascending scale of severity and a progressive series of clinical pictures, are chosen for noting in detail, after which the author gives an interesting résumé of the opinions as to the anaesthesia of leprosy enunciated by such authorities as LE DANTEC,

Jeanselme, P. Manson, Brissaud and Déjerine. The conclusions of Dr. Cussec himself are as follows:—

"It would appear, then, that the anaesthesia of leprosy is caused by a degenerative neuritis of bacillary origin spreading, on the one hand, superficially along the skin and affecting more and more of the nerve terminations in its course, and, on the other hand, taking a deep course by ascending the nerve fibres themselves, finally reaching the roots and gaining the posterior horns of the spinal cord. At this level, the lesions affect that portion of the grey matter of which the cutaneous 'projection' is that of the corresponding posterior nerve roots. As a matter of fact, numerous lepra bacilli have been demonstrated in these situations, where their presence has led to sclerosis and the formation of fibrous nodules, with ultimate atrophy of nerve tissue."

S. L. C.

Agricola (Ernani). **Da puncção ganglionar no mal de Hansen.** [Gland Puncture in Leprosy.]—*Thèse de doctorat, passée devant la Faculté de Bello-Horizonto, État de Minas Geraés, Brésil.* 80 pp. Typ. Ariel, Bello Horizonto.—[Summarized in *Bull. Inst. Pasteur.* 1921. Mar. 30. Vol. 19. No. 6. pp. 195–196.]

The author describes an investigation in which the lepra bacillus was sought for in the superficial glands of 19 patients suffering from the disease. The findings were positive in no less than 12 of these cases. Further, an examination of gland pulp from two other persons free from symptoms or signs of the disease, but closely associated with known or suspected cases, led to the finding of the bacilli, and thus rendered possible a diagnosis at a moment when clinical examination afforded no information.

Impressed with the importance of these observations, the author suggests that every suspected case should be submitted to this test. He classifies cases of leprosy into "glandular," "nervous," "cutaneous" and "mixed."

[Although most students of leprosy will agree that this classification is rather premature, the work on which the author bases his paper is highly interesting and suggestive, and confirmation by other observers will be awaited with interest. See Leboeuf, this *Bulletin*, Vol. 1, p. 191.]

S. L. C.

Bruusgaard (E.). **Bidrag til Kjendskapet av den saakaldte tuberkuloide lepra.** [Contribution to the Knowledge of the So-called Tuberculoid Leprosy.]—*Norsk. Mag. f. Laegevidensk.* 1921. Vol. 82. No. 5. p. 359.

The case was that of a woman, 65 years of age. She had a skin affection, chiefly on the distal part of the left hand and foot, consisting of multiple disseminated patches of reddish-brown infiltrated papules, soft in consistence, which on pressure with a glass spatula left yellowish-white foci. It much resembled multiple disseminated lupus. The W.R. was negative. In addition she had marked atrophy of the muscles of the left hand, with consequent paresis. The nails were thin and flat. The ulnar nerve was felt to be thickened. She complained of shooting pains in the hand. X-ray examination showed marked atrophy and calcification. Although the diagnosis of the skin lesion was difficult, paresis of the hand and swelling of the ulnar nerve pointed to leprosy. It did not conform to the maculo-anaesthetic

type, but with its lupus-like appearance it was undoubtedly a case of JADASSOHN's tuberculoid leprosy. In sections of the skin undoubted leprosy bacilli were not to be found, only a granular acid-fast rod here and there in the protoplasm of the connective tissue cells. Tuberculosis was excluded by an injection of tuberculin, and the inoculation of guinea-pigs with an extract of the skin. A microscopic examination of the ulnar nerve showed the same granulation tissue as in the skin, but no typical giant cells or necrotic foci. This supplements JADASSOHN's description of this form of the disease. Although the patient received no treatment she got much better. In the course of two years no new lesions of the skin appeared and the old ones were partially or wholly absorbed. The neurological examination gave about the same result as when she was admitted to hospital. Her general condition was excellent. The tuberculoid form of leprosy is perhaps the form *par excellence* in which the patient most easily overcomes the disease, and possibly in a shorter time than is the case with the maculo-anaesthetic type, to which it is most nearly related. On account of its characteristic clinical picture and its rarity it is advisable to look upon it as a distinct variety of the disease; otherwise it will be easily overlooked in practice. This is the first case of tuberculoid leprosy reported in Norway.

E. E. Atkin.

KOHDA (K.). **On the Cultivation of** *Bacillus leprae.*—*Kitasato Arch. Experim. Med.* 1921. Mar. Vol. 4. No. 2. pp. 141–158; and *Sei-i-Kwai Med. Jl.* 1921. Mar. Vol. 40. Nos. 1–2. (Whole Nos. 456–457.) pp. 1–15.

This work consists of two parts, the first being concerned with the morphological and cultural characters and the results of animal inoculation with the Kedrowski bacillus. A strain of this organism, brought to Tokyo from Breslau by Prof. DOHI, was carefully tested by the author, who records his results in the paper now under review. In the main, the results obtained are comparable with those recorded by others, but Kohda found no evidence of the formation of streptothrix forms in his subcultures, and the acid-fast character was always retained at the same level. Inoculation into guinea-pigs, rabbits, rats and mice showed that, if given in sufficient quantity, the organism was capable of bringing about pathological conditions of a subacute order, and, in some instances, even death. In none of the animals were symptoms like those of leprosy produced, except that in the case of one of the rats there was loss of hair. The second part of the paper discusses experiments carried out to test the antibody content of the blood of lepers for antigen derived from Kedrowski cultures. It was shown that agglutinins were present in four out of ten cases in low dilution. but avian tubercle bacilli were also agglutinated. In complement fixation tests, 33·3 per cent. of the sera were positive both to the Kedrowski and the avian strains. The work in no way points to the Kedrowski bacillus as the cause of leprosy.

S. L. C.

HOOPER (Philip). **The Symptomatology and Treatment of Leprosy.**—*Jl. Trop. Med. & Hyg.* 1921. May 16. Vol. 24. No. 10. pp. 137–140. With 1 chart in text.

This is a good clinical paper, in which an interesting account is given of the frequency of various symptoms in a series of 41 cases under

observation at Makogai Leper Asylum in Fiji. Important items in this analysis are the positive findings in 21 out of 41 single examinations of nasal mucus for acid-fast bacilli, the finding of 23 from the total with glandular enlargement, the observation that nerves may be enlarged without any marked symptoms of modification in sensation or power, and the discovery of anaesthetic patches independent of macules or nerve lesions in 30 cases. The author finds it possible to give crude chaulmoogra oil intravenously in doses of 15 minims daily for six days a week for months on end without ill effects. Of 40 cases so treated, only two failed to derive benefit from the drug. There is a rise of temperature and pulse after the injections, lasting about eight hours. A table is given showing the total amount of oil given, the number of injections, the weight at the commencement of treatment and at the end of the year, and the age and type in each of the 40 patients treated. It is to be noted that nine of these cases were at an age when weight is usually gained through natural growth, except in very advanced cases. Of the 40 patients concerned, 25 gained weight, 11 lost and 4 remained stationary. It would be necessary to know much more about each individual case before drawing any conclusion from this table, but the paper is written with knowledge and conviction, and we get the impression of a very successful effort in treatment more from the article itself than from this table of weights.

S. L. C.

Jeanselme (E.). **Rapport sur une nouvelle méthode de traitement de la lèpre, instituée par le Dr. Harry T. Hollmann, présenté au nom de la Commission permanente de la lèpre.**—*Bull. Acad. Méd.* 1921. Mar. 29. Vol. 85. No. 13. pp. 393–395.

The author, in this paper, brings to the notice of the Académie de Médecine a memoir by M. Marques, French Consular Agent at Honolulu, received through the Minister of Public Instruction, in which the work of Dr. Harry T. Hollmann in the treatment of leprosy by means of the fatty acids of chaulmoogra oil and their ethyl esters, already reviewed in this *Bulletin* [Vol. 15, p. 272], is described, together with a summary of the method of preparation and use, and the results obtained. It is curious to find all reference to the investigations of Sir Leonard Rogers and his co-workers omitted in the presentation of a report on what is described in the title as a new treatment. The paper is written with great lucidity, and is to be recommended to those who desire to find a clear description of technique.

S. L. C.

Horta (Parreiras). **Prophylaxia e tratamento da lepra.** [Prophylaxis and Treatment of Leprosy.]—*Rev. Med. Chirurg. do Brazil.* 1921. Feb. Vol. 29. No. 2. pp. 67–71.

The author, aided by two friends, the chemists Paulo Ganns and Dias da Cruz Netto, who prepared for him the ethyl esters of the fatty acids of chaulmoogra oil, has tried these preparations in the treatment of leprosy, with very satisfactory results. The preparation produced by these workers on the lines indicated by the researches of Hollman and Dean, has been called by them "Chaulmougrol," and would appear to be, on the whole, more powerful than that of the American scientists, and to lead to reactions of a characteristic kind. Its use is followed by cure in a high proportion of cases, lesions drying up and the

bacilli disappearing from the discharges and from the tissues. Speaking of the all-important question of prophylaxis, the author commends the zeal with which the Public Health Service is dealing with this problem, but he hints that the proposed provision of leprosariums and leper colonies will be very costly, and that it will hardly be possible to afford more than the construction of two or three such institutions in the next few years; probably not enough to accommodate the native lepers, the disease being very common in Brazil. He points out that the discovery of a specific cure places a valuable prophylactic measure at the disposal of the Sanitary Authorities, as the treatment and cure of cases at dispensaries will cheaply and surely eliminate foci of infection.

S. L. C.

DE MELLO (Froilano). **Traitement de la lèpre.**—*Presse Méd.* 1921. Oct. 29. No. 87. p. 861.

In this memorandum de Mello reiterates several of his conclusions already published (see this *Bulletin*, Vol. 17, p. 282) and adds certain details as to cases treated since his last communication. He speaks with optimism as to the advances now being made in the treatment of leprosy with the unsaturated fatty acids of chaulmoogra oil and also hints that there is a greater field for work in connection with K. K. CHATTERJI's investigations into the properties of oil of "Nim," the indigenous name for the margosa tree or "Cuddo Nimb" (Hindustani). He strikes a note of caution as to the use of the word "cured" for cases diagnosed leprosy in whom, under treatment, the lesions have totally disappeared and the lepra bacilli ceased to be demonstrable in the sites where the lesions existed or even in the nasal secretion after the use of iodides; but the fact that this state of apparent freedom from disease has been known to last, already, for four years, and that the ex-patients have reassumed their places in society and their previous vocations, gives good ground for the hope that such "cures" are in every way real.

De Mello finds that he can use, by intramuscular injection, solutions of gynocardate of soda up to 4, 5 and even 6 per cent. He commences with a dose of 5 cc.—but of what exact strength, 4, 5 or 6 per cent., is not stated—and finds that he can raise the dose to 12 cc. with safety. He is of opinion that the gynocardate pills used *per os* by ROGERS are of no value, the injections constituting the effective part of the treatment; and so he confines himself to the latter. As mentioned in our previous summary, the author finds reason to think that the fragmentation of the lepra bacilli noted by ROGERS in the nodules of cases treated with the gynocardates is of little or no significance, since similar appearances may be noted in the same proportion of counted bacilli in treated and untreated cases. He reserves the results of treatment by "Oil of Nim" for a further communication.

S. L. C.

DEAN (Arthur L.) & WRENSHALL (Richard). **Fractionation of Chaulmoogra Oil.**—*Public Health Rep.* 1921. Apr. 1. Vol. 36. No. 13. pp. 641–660. With 4 text figs.

In a brief introduction the authors review the history of chaulmoogra oil and its derivatives. Formerly thought to be the product of *Gynocardia odorata*, the oil was shown by POWER and his co-workers of the Wellcome Chemical Research Laboratories to be in reality

derived from the seeds of *Taraktogenos kurzii*, while the oils from two closely allied species of *Hydnocarpus* were practically identical. Power showed that Taraktogenos and Hydnocarpus oils contained a new type of fatty acid, and two acids of this series were isolated and investigated. "Chaulmoogric acid melts at 68°, has an iodine value of 90·1, and shows a specific rotation of + 56; hydnocarpic acid melts at 59°, possesses an iodine value of 100·2°, and gives a specific rotation of + 68·1°." In the search for a suitable form for therapeutic use, the mixed fatty acids of chaulmoogra oil, being solid at ordinary temperatures, were not convenient; the soluble salts were regarded as dangerous for intravenous use, but the ethyl esters of the fatty acids, prepared by Dean, were found to be thin liquids suitable for intramuscular injection, being readily absorbed and well tolerated.

The results obtained with these preparations were so favourable that it became very important to ascertain whether any one of the four fractions of fatty acids used was conspicuously superior to the others, and it was therefore necessary to obtain these fractions separately in considerable quantity. The methods employed to obtain sufficient quantities of these acids in a state of purity are described in the paper under review. An attempt was first made to separate the fatty acid by crystallization from alcohol. The technique employed is described in detail, but the method was not found satisfactory and was abandoned. Attention was now turned to the separation of the fatty acids by means of barium acetate. By this means, both chaulmoogric and hydnocarpic acids were obtained pure, but, although capable of improvement, the method did not afford hopes of the production of the acids in sufficient quantities. Better results were obtained by fractional distillation of the ethyl esters under high vacuum, and this method, though for the moment abandoned in favour of a more productive technique, is promising, and will be further investigated in the future. The best results were obtained by the fractional distillation of the fatty acids themselves *in vacuo*. For the exact technique evolved the original paper should be consulted. The work has been rendered more difficult through the inferior quality of the chaulmoogra oil placed on the American market, the supply available being a dark reddish-brown oil, instead of the clear amber-coloured oil obtainable in 1919, and being found to contain the normal amount of chaulmoogric acid, but only about one third of the hydnocarpic acid formerly recovered from the lighter-coloured oil. Even from low grade oil, the method advocated will give at least 50 gms. of pure hydnocarpic, and 100 gms. of chaulmoogric acid from 1,000 gms. of mixed fatty acids.

S. L. C.

Ghosh (Sudhamoy). **Chemical Investigation in Connection with Leprosy Inquiry. Part II.**— *Indian Jl. Med. Res.* 1920. Oct. Vol. 8. No. 2. pp. 211–215.

This article, a continuation of a research by the author on the chemistry of chaulmoogra oil and some other oils closely related thereto, though of great importance, does not lend itself well to summary, as the work is of a highly technical nature and already condensed to the smallest possible space. Readers who desire to inform themselves as to the chemistry of the preparations of chaulmoogra oil are therefore referred to the original. Dr. Sudhamoy Ghosh

has also investigated cod-liver oil, and he thinks that the unsaturated fatty acids of cod-liver oil and of soya bean open up a field of research likely to yield fruitful results. It is interesting to note that, although WALKER and SWEENEY [see this *Bulletin*, Vol. 17, p. 277] found sodium morrhuate to be relatively inert in bactericidal power for acid-fast organisms, LINDENBERG and RANGEL PESTANA [see pp. 390–391 above] have found it to prevent the growth of some of the commoner bacilli of this group, and we have the researches of ROGERS, Ghosh and others to indicate that the morrhuate is of real value in the treatment of leprosy. It is to be hoped that further experiments *in vitro* with derivatives of cod-liver oil may be tried in order to settle this point.

S. L. C.

McDONALD (J. T.) & DEAN (A. L.). **The Constituents of Chaulmoogra Oil effective in Leprosy.**— *Jl. Amer. Med. Assoc.* 1921. May 28. Vol. 76. No. 22. pp. 1470–1474.

From 1st January, 1920, to the middle of March, 1921, practically all the patients at the Kalihi Hospital have been receiving the "standard treatment" described by the authors in their previous publications [see this *Bulletin*, Vol. 17, p. 281]. Of these patients, 50 per cent. have been "paroled" after recovery. Most of the 87 patients now in hospital are comparatively recent arrivals. As the authors put it, "It would appear that we have methods in hand for holding the disease under control." In addition to the steady use of their standard method, the authors have also tried out several lines of investigation, of which three are as follows :—

1. What is the relative value of the oral administration of chaulmoogra compounds as practised in our standard treatment? The answer to this question, obtained by treating ten patients by injections alone without any oral administration except of a tonic medicine, is to the effect that the injections are just as effective by themselves and that the oral administration is merely waste of valuable medicine.

2. What are the therapeutic agents in chaulmoogra oil? By means of specially devised methods, of which the details are not given in the paper, but have appeared elsewhere (*Jl. of Amer. Chem. Soc.*, Vol. 42, 1920, pp. 2626–2645), considerable quantities of pure crystallized chaulmoogric and hydnocarpic acids were prepared, and these were converted into their ethyl esters, the latter being distilled to get them into the highest state of purity. Of ten patients, five were treated by the ethyl ester of chaulmoogric acid and five by that of hydnocarpic acid. On the whole, the latter gave the better results, but both were highly satisfactory. The authors conclude that "on a basis of these results" it may be asserted that "acids having the peculiar structure and properties of chaulmoogric and hydnocarpic acids will arrest human leprosy." By means of further research they were able to obtain a preparation with an iodine number of about 125, that is, about 35 points higher than the iodine value of chaulmoogric acid. The indications are that this is a mixture of chaulmoogric acid with a compound of the same nature as the latter but more highly unsaturated. With this preparation a group of five patients were treated, of whom three improved with unusual rapidity, one was soon paroled, and the fifth, an old man who was very irregular in taking injections, was allowed to drop out of the series.

3. Experiments proved that iodine is not an essential item in the treatment.

It would appear that something like 10 per cent. of the patients are unable to take injections continuously owing to a tendency to break out into swellings, "which may perhaps be a form of toxaemia." These, though not serious, interrupt the course of treatment, and investigation is desirable with a view to their prevention. This paper is a highly important one and should be read in the original.

S. L. C.

ROGERS (Leonard). **Chaulmoogra Oil in Leprosy and Tuberculosis. The Successful Treatment of Leprosy by Injections of Soluble Preparations of the Fatty Acids of Chaulmoogra and Other Oils and its Bearing on the Tuberculosis Problem.**—*Lancet.* 1921. June 4. pp. 1178–1180.

In this paper Sir Leonard Rogers summarizes the work done by him and his co-workers in India on the use of the new preparations of chaulmoogra oil. After a short review of the history of these preparations in the treatment of leprosy he explains why the hydnocarpic acid was considered to be probably the most active constituent of the oil, and explains how, through later observations, it has been found that active preparations can be made from the whole of the fatty acids of these oils without the labour of fractionation. He then passes on to the use of sodium morrhuate in tuberculosis, but confines himself to a very brief reference to the work on this substance and on unsaturated fatty acids of soya bean, which he thinks have every prospect of successful use in tuberculosis as well as leprosy in the future.

Turning to results, he quotes his contributions to the Calcutta Leprosy Conference [see this *Bulletin*, Vol. 17, p. 286], and states that the duration of treatment should be a year or more, if possible. In cases treated with the chaulmoogra acid preparations for a year or longer no less than 9 out of 13, or 65 per cent., showed complete disappearance of the lesions. Of 20 cases treated with sodium morrhuate 3 were slightly improved, 12 greatly improved, and in 5 the lesions had disappeared. The after-results of 26 gynocardic cases and 9 cases treated with morrhuate had been followed up for another year subsequent to an earlier report. In the former 5 only had relapsed, all being patients who had discontinued treatment against advice. Some of these subsequently cleared up under further treatment.

Sir Leonard lays stress upon certain statistics which seem to show that there is an especial susceptibility to leprosy infection during the first two decades of life as compared with later age.

Turning once more to treatment, the author states that the results in tuberculosis are variable, but on the whole, in his opinion, good. He further states that the treatment was harmless in the cases that had come under his observation in 1919. In this connection the results recorded by LINDENBERG and RANGEL PESTANA should be borne in mind [see pp. 390–391 above], as in their hands unsaturated fatty acids have proved to give rise to very definite reactions in cases of pulmonary tuberculosis; and there would appear to be some reason to think that caution must be used in applying the results obtained in leprosy to tuberculous infections, caused by a much more toxic type of acid-fast bacillus.

S. L. C.

Cawston (F. G.) ; Robertson (Wm.). **Treatment of Leprosy.** [Correspondence.]—*S. African Med. Rec.* 1921. Aug. 13. Vol. 19. No. 15. p. 300.

Dr. Cawston, whose previous notes re the treatment of leprosy by means of " oscol stibium " have been reviewed in this *Bulletin* [Vol. 17, p. 284], writes to point out that, in view of his findings that the drug should be " pushed until toxic symptoms, such as diarrhoea, have shown themselves," the failure of smaller doses at Robben Island, as reported in the lay press, is possibly to be explained in terms of insufficient dosage. He suggests, too, that the known fact that pathogenic organisms can become antimony-fast may have a bearing on these cases. He has recently advocated the use of Thiarsol, a colloidal preparation of arsenic and sulphur, to be given after a preliminary course of treatment with antimony in leper patients.

Dr. Robertson briefly reports a case of leprosy treated initially with " 606," the Wassermann reaction being positive, and subsequently with intravenous inoculations of tartar emetic, from one-fifth to one-fourth of a grain. The results were excellent. The solution used was one containing a grain of tartar emetic to the drachm, a piece of camphor being added to prevent the growth of moulds.

S. L. C.

Roussel (J. N.). **Two Cases of Leprosy cured by Anthrax Vaccine.**—*New Orleans Med. & Surg. Jl.* 1921. Oct. Vol. 74. No. 4. pp. 250–252. (Discussion, pp. 253–255.)

After a few preliminary remarks as to the complete failure of all previous attempts at the treatment of leprosy from the time of Moses to the Hansen period—but without any reference to the work of Rogers, of McDonald, of Muir and others with the new chaulmoogra oil derivatives—the author proceeds to describe two cases in which, under his treatment by injections of the " liquid anthrax vaccine of H. K. Mulford Co., for the immunization of animals against anthrax," a cure was rapidly obtained. In one of these cases acid-fast bacilli were demonstrated, but in what situation is not stated. In the other, attempts to find acid-fast bacilli proved fruitless. The clinical findings are recorded in such a scanty manner that it is quite impossible to judge whether the patients suffered from leprosy or not, but the author is not in any doubt upon this score. One of the cases came under notice in February and the other in March of 1919, so that not quite three years have elapsed since they were first seen ; nor is there any statement that they were kept under observation after the summer of 1919.

In the discussion which followed the report of these cases at the New Orleans Medical Society, Dr. H. E. Menage quoted recent work and sounded a note of caution as to accepting the bacillus-negative case as one of leprosy, but, on the whole, the communication was well received. In this *Bulletin* the paper is noted merely to serve as an example of how very uncritical may be the attitude of the general practitioner of the effects of measures employed in treatment.

S. L. C.

Wayson (James T.). **The Value of Iodin in the Treatment of Leprosy.**—*Arch. Dermat. & Syph.* 1921. Mar. Vol. 3. No. 3. pp. 248–249.

Wayson, reverting to a method of treatment used by Danielson and others in the period between 1860 and 1890, has tried " potassium

iodid" in leprosy and claims very good results, more especially in the nodular variety. With doses gradually increased to between 100 and 200 gr. daily, large nodules have been found to break down into ulcers and finally heal with but little scar tissue; while sensation, though lost, has been restored. These beneficial results have been obtained at the cost of considerable suffering to the patient, excruciating neuralgic pains and loss of sleep being endured, with the compensation of a decided improvement later on. The author has also tried iodine in the form of sajodin, a 32 per cent. iodine powder, in doses gradually increased to 20 gr. three times a day, and, later, iodalbin, a preparation containing 22 per cent. of iodine, with even better results. Lugol's solution, in increasing doses to 20 drops three times a day, has also been used with success. A curious development of this method, described by Dr. Wayson, consists in freezing the skin lesions of patients under iodine treatment by means of carbon dioxide snow to produce a large bulla, the contents of which are removed for use in treatment. The fluid from the bulla is regarded as containing iodine and is described as an "iodinized autogenous serum," being reinjected into the patient. Skin lesions containing large numbers of bacilli are selected for this purpose. The note is brief, and no details of the progress of treated or control cases are given, nor are the numbers of patients dealt with specified.

S. L. C.

LINDENBERG (Adolpho). **Da prophylaxia geral á prophylaxia da lepra.**—*Bol. Soc. Med. e Cirurg. de S. Paulo.* Brazil. 1920 & 1921. Oct.–Feb. Vol. 3. (2nd series.) Nos. 8–12. pp. 348–350.

The author defines certain landmarks in the history of preventive medicine, from the methods in general use in the Middle Ages, consisting almost exclusively of compulsory isolation, to which was joined, later, notification, on through the stage of "specific prophylaxis," that dawned with Jennerian vaccination, to the period of "measures of general hygiene" based on the discovery of the microbic origin of infectious disease. The disappointing results of these general measures led back to a revival of the principles of specific prophylaxis, now extended to a whole group of diseases by the use of active immunization with killed or attenuated cultures of the germs concerned. This line of activity has been supplemented by the introduction of "therapeutic prophylaxis," first used by KOCH in the attempt to diminish malaria by quininization of cases in order to reduce sources of infection to the insect host, and typically employed in the campaign against syphilis by the disinfection of cases by EHRLICH'S salvarsan. The author, for the suppression of leprosy, a disease of numerical as well as clinical importance in Brazil, advocates what he describes as "indirect therapeutic prophylaxis," that is to say, the treatment of patients at dispensaries and the admission of advanced and infective cases to sanatoria; in fact, the measures now employed in most tuberculosis schemes. This suggestion assumes increased importance in view of the fact that leprosy must now be regarded as a much more therapeutically curable disease than tuberculosis. It is quite possible that the dispensary treatment of early cases by means of the new preparations of chaulmoogra oil may yet prove to be a highly practical means of dealing with this disease.

S. L. C.

MARCHOUX (E.). **Une nouvelle maladie à bacilles acido-résistants qui n'est ni la tuberculose, ni la lèpre.**—*Bull. Acad. Méd.* 1921. Mar. 8. Vol. 85. No. 10. pp. 317–319.

The patient in question was a native of Haiti, aged 23 years, admitted to the Hôpital Pasteur under the care of Dr. Louis MARTIN and Dr. VEILLON, through whose kindness the author was enabled to investigate the case. Believed to be suffering from leprosy, the patient, nevertheless, presented certain peculiarities, since there was an absence of zones of anaesthesia and no trace of nodules. The skin, however, showed curious pigmented spots on the face and limbs, these being the result of ulcers which had followed upon bullous eruptions. Over these spots sensibility to pin-pricks and to heat was unaltered. The conjunctivae and mucous membranes were quite healthy. The diagnosis of leprosy would have been difficult to explain had it not been for the fact that, in films from the nasal membranes and from the serous fluid of the vesicles, colonies or masses of acid-fast bacilli were constantly found. Eventually the case came to the post-mortem table, the patient having died of a purulent pleurisy associated with streptococci in pure culture. It was then that Dr. Marchoux was able to ascertain for certain that he was dealing with an acid-fast organism differing from both the tubercle and the leprosy bacillus.

In films from all the organs, but particularly from the spleen, could be found masses of bacteria arranged in spherical globi and surrounded by resistant gelatinous material, or held together in little drops of fatty substance. These masses consisted of very fine short organisms arranged anyhow, and offering in this a contrast to lepra bacilli, which are usually arranged in bundles of parallel germs. Stained with warm carbol-fuchsin, they resisted decolorization by acids or alcohol, but were readily decolorized by xylene, toluene, acetone, ether and chloroform. After decolorization by means of wax solvents, the bacilli could no longer be stained with fuchsin, a character in itself sufficing to distinguish them from Koch's and Hansen's bacilli. The dimensions, shape, and arrangement in masses resembling little collections of red powder, and the way in which the individual elements disperse when the containing vacuole is ruptured, suffice to justify for the new germ the name, proposed for it by its discoverer, of *Mycobacterium pulviforme*. It is an organism of very small size, its dimensions being $0 \cdot 5–1\mu$ in length by $0 \cdot 25\mu$ in breadth. The "masses" attain dimensions of from $2–20\mu$. The masses could be found in the enlarged spleen, in the liver, where they were less numerous, and in the bronchial and mesenteric glands.

The case is one of extreme interest, and excites keen speculation as to its real nature. It is much to be regretted that the description fails to provide information as to the results of cultural and inoculation experiments, or of histological observations.

S. L. C.

REVIEWS.

ROGERS (Leonard) [C.I.E., M.D., F.R.C.P., F.R.S., I.M.S. (Retd.)]. **Bowel Diseases in the Tropics: Cholera, Dysenteries, Liver Abscess and Sprue.**—pp. xvi + 475. With 16 figs., 10 plates, 2 charts and 3 diagrams. 1921. London: Oxford Medical Publications. Henry Frowde and Hodder & Stoughton, The Lancet Building, 1 & 2, Bedford Street, Strand, W.C.2. [Price 30*s.* net.]

It was a happy idea of Sir Leonard Rogers to combine in one volume the substance of his two earlier works on cholera and dysentery. This new volume on "Bowel Diseases in the Tropics" has given him the opportunity of revising both of them in accordance with his further experiences at Calcutta during the last eight or nine years. So assiduously has the author devoted himself to the study of these diseases that any opinions that he holds regarding them demand very careful consideration.

The clinical descriptions are excellent. The account of the symptoms of cholera could never have been written by anyone who had not had very great experience of this disease, and observed it in the true scientific spirit.

The title of the book is perhaps somewhat ill-chosen. There are very scanty allusions to any parts of the tropics other than India, although, especially of late years, a large amount of work on bowel diseases has been done in other countries. But the fact that Sir Leonard Rogers confines himself almost solely to his own observations does not diminish materially the value of the monograph. It represents the mature opinions of one who is entitled to write with authority on the subjects dealt with.

A more pertinent criticism is that the author deals almost entirely with the diseases as seen at the Medical College Hospital, Calcutta, where the conditions are quite exceptional for the tropics. For instance, in the case of cholera, when one sees described, as practical guiding lines for treatment, such procedures as the estimation of the specific gravity of the blood, of its chlorine content, the blood pressure, and differential leucocyte counts, one cannot help thinking that to insist on such refinements (well enough in their proper place—in a well-equipped hospital, with plenty of trained assistants, and where only a few cases are treated daily) is apt to oppress the mind of the unfortunate, over-worked medical officer, who has to deal with an epidemic of the disease under the usual conditions.

A somewhat undue proportion of the book is devoted to cholera, of which a very full description is given. The historical account, which is reproduced from the earlier book, is excellent, and of much more than sentimental interest. Such a story as that of the gradual acquisition of scientific knowledge of the nature of cholera must prove decidedly stimulating to anyone who desires to increase it.

There are many old, and many new, Indian reports on outbreaks of cholera: little reference is made to these. The conditions described so graphically by the old Anglo-Indian medical officers may be seen, with very little change, to-day. MURRAY (as quoted by HIRSCH), in his General Report for the Upper Provinces for 1868, says, describing the pilgrims hastening to their homes in wild flight and panic, on the appearance of the disease at Hardwar: "The moving mass crowded the road in a continuous stream for nearly a week at Meerut, where I remained to watch the course of the disease. This pilgrim stream carried with it cholera, which lined the road with victims, whose funeral pyres studded the surrounding fields, or whose bodies were thrown into the canal, or collected by the police and buried. The disease was communicated to the neighbouring towns and villages, and the pilgrims carried it with them to their homes over the whole of Hindustan."

It is, indeed, in circumstances such as these, that cholera has to be treated by most medical men. But Sir Leonard only refers to this in the most general terms.

The statistics given of the result of treatment in Calcutta are stimulating and of value, but one must remember that the conditions there are unusually favourable. It would have been of more general interest and of more practical advantage to give the results of treatment in the epidemic outbreaks at the big Indian bathing fairs, where Rogers's methods were followed by others than their author.

I cannot share Sir Leonard Rogers's fondness for statistical tables. In any case, as he himself admits, the figures given under the different diseases, in the Indian mortality tables, are notoriously of very little value, with the exception of those referring to the Army, the Jails, and a few other institutions under the direction of competent medical officers. Such a table, therefore, as that given on page 37, which professes to give the monthly provincial cholera death-rates for the whole of India, and which the author discusses at some length, cannot be taken very seriously by anyone who has lived in that country. These figures are compiled, in the first instance, by the village "chaukidar," or watchman, an ignorant rustic, whose remuneration is about four rupees a month, and who arranges his figures in accordance with local expediency rather than with the facts, even as he interprets them.

In the paragraphs on mortality from cholera, the figures of the annual reports of the Sanitary Commissioners with the Government of India, for British and Native troops, and the jail population, are quoted; but only those for the decade ending in 1908 are given. As these are the only Indian statistics derived from the returns of qualified medical men, they are of considerable importance. It would have enhanced the value of the book if use had been made of the later reports referring to the years after the hypertonic saline treatment came into general use.

The basis of Rogers's treatment is the effect of hypertonic saline injections upon the blood-condition. In "Cholera," a table is given showing these results in twenty-four cases. It is disappointing to find this same table here reproduced. Twenty-four cases are surely a very small foundation for so large a superstructure. Of course, I do not suppose that Sir Leonard Rogers has no notes of other cases bearing on this matter, but I think that he should have published them here.

The only important addition to the section on the blood-changes in cholera is a rather brief paragraph, reinforced, it is true, by two tables, on the Reduction of the Alkalinity of the Blood in the disease and its relationship to post-choleraic uraemia. Sir Leonard fully confirms the opinion of SELLARDS on this subject, and carries the matter considerably further as regards its practical applications.

The chapter on the diagnosis of cholera is very good. To those who have only a book knowledge of the disease it may seem that there can be no great difficulty about this; but Sir Leonard very rightly emphasises the fact that a good many cases present themselves in which an immediate diagnosis is not possible.

In the reviewer's opinion much of the rather elementary bacteriology of cholera might have been omitted.

In the chapter on treatment the author says that the subcutaneous injection of saline solutions "has been most frequently relied on in the treatment of cholera collapse during recent years in India." This, I think, must have been copied from the 1911 edition, and must have escaped the author's notice. Since that book appeared, practically every British and Indian medical officer has treated his cases according to Sir Leonard's recommendations.

Table No. XX, on page 189, is of the highest interest. It shows what can be done in favourable circumstances and by skilful hands. It is indeed a triumph for Sir Leonard Rogers to be able to record the results of treatment at the Calcutta Medical College Hospital during the years 1915–19, when hypertonic saline and alkaline injections combined with the internal administration of permanganates were used. During this period 1,429

cases were treated, with only 298 deaths. In other words, over 79 per cent. of the patients recovered.

The most important new detail, in the treatment now adopted, is the use of an alkaline solution, designed to counteract the "acidosis" of the blood in severe cases of cholera. The solution used is as follows :—

Sodium bicarbonate	160 gr.
Sodium chloride	90 gr.
Water	1 pint.

Rogers now uses one pint of this alkaline solution in every case requiring an intravenous injection.

He rightly lays stress upon the high febrile reaction which frequently follows large saline injections, and advocates that they be given at a temperature considerably below that of the blood if the rectal temperature is high. If the rectal temperature exceeds 102° F. he does not warm the saline solution at all.

The chapters on dysentery are very good ; and the fine coloured plates of the bowel lesions in amoebic and bacillary dysentery, which first appeared in the original edition, are reproduced. There are many alterations and additions, though most of the tables on liver abscess are copied from the old book. There is an excellent account in Chapter VIII of the methods used for demonstrating the presence of *Entamoeba histolytica* in the stools ; and the chapter on the treatment of amoebic dysentery is considerably amplified. Most readers would have welcomed a somewhat fuller account of bacillary dysentery ; those also of Hill diarrhoea and sprue are rather inadequate.

The book shows evidence of rather hasty proof-reading. Besides some obvious misprints, one is surprised to find the old mis-spellings—"Tricomonas, Heterotrica, Mesneli"—which disfigured the earlier books, reproduced. In the preface, the *Tropical Diseases Bulletin* is referred to as the "Bulletin of Tropical Medicine."

"Bowel Diseases in the Tropics" will assuredly enhance the high reputation so deservedly enjoyed by Sir Leonard Rogers, and will be an indispensable *vade mecum* to all practitioners in the tropics. In such a vast field there must necessarily be certain points concerning which individual experiences and opinions differ. Sir Leonard Rogers's name will be written large for all time in the history of cholera and dysentery ; but even to one with his great experience it is not given to write "Finis" to the account of any human disease.

H. J. Walton.

Kofoid (Charles A.) & Swezy (Olive). **The Flagellate Infections of the Human Digestive Tract.** — *Nelson's Loose Leaf Encyclopedia of Living Medicine.* Vol. 5. (Chap. XX.) pp. 365–398. With 6 plates. 1921.

The flagellates of the human alimentary canal form a small but interesting group. Much has already been written about them, but in recent years few attempts have been made to sift and summarize our scattered knowledge of the group as a whole. Consequently, the present article, which is evidently designed as a comprehensive survey of the whole subject, will be eagerly studied by many workers—more especially as the senior author, Professor Kofoid, is already well known as the author of numerous publications on the flagellate Protozoa. During the Great War, moreover, he occupied a leading position among the workers engaged in studying intestinal protozoal infections in the American troops, and he has therefore had recent opportunities of obtaining a considerable first-hand knowledge of the subject. The reader will thus feel justified in approaching the present article with great expectations. He will look to see many a knotty point unravelled, and many a dark place illuminated by the light of a ripe zoological experience.

The authors have here attempted something more than the title of their work implies : for in addition to describing the intestinal and oral flagellates of man, they treat of the trichomonads recorded from the vagina and the lung, and of a variety of coprozoic organisms. Their reasons for including such forms are not, at first, easy to guess. But perusal of their work makes it clear that they are still in some doubt as to which of the flagellates described from man are truly coprozoic, and which are not ; and their apparently catholic interpretation of "the digestive tract" is, perhaps, explained by the following passage from the Introduction. "It is . . . to be expected from their structure and from our knowledge of their occurrence and distribution in other vertebrates, that the principal flagellate parasites of the intestine may have more generalized capabilities of tissue invasion than has hitherto been suspected or detected." Those who know what has already been "suspected or detected" in this line may feel somewhat staggered at the vista opened up by this remark : but they will doubtless, like the reviewer, take comfort from the thought that these dire discoveries have not, as yet, been made.

The brief introductory remarks are followed by a section dealing with the "Technique of Examination." This contains little that will be novel to readers of the *Bulletin*—except, perhaps, the modified eosin-iodine solution which is recommended for use in the examination of stools. The modification advocated consists in the employment of physiological saline solution—instead of water—for making up and diluting the stain : which seems to be merely the result of a misconception regarding the use of "physiological" solutions generally. It may be noted, further, that "formalin" (strength not stated) for "5 minutes only" is given as an alternative to Schaudinn's fluid as a fixative for wet coverglass films of faeces. It is hard to believe that anybody who has tried this method for fixing protozoal cysts in human faeces could ever recommend it to others.

In the descriptive sections which ensue, attention is paid to the morphology and nomenclature of the following flagellates, together with discussions of their occurrence and pathogenicity, and of the diagnosis, pathology, and treatment of the various infections :—*Giardia enterica* (Professor Kofoid's new combination of names for *Giardia intestinalis*—resting, apparently, upon a mistake made many years ago by Diesing) ; "*Craigia*," with two species ; *Chilomastix davainei* (better known as *Ch. mesnili*) ; *Trichomonas hominis, T. vaginalis, T. buccalis,* "*T. pulmonalis* Schmidt," and "*Pentatrichomonas ardin delteili*" ; "*Tetrachilomastix intestinalis*" ; *Tricercomonas intestinalis* ; *Embadomonas intestinalis* ; "*Ditrichomastix hominis*" ; *Diplocercomonas soudanensis* ; *Enteromonas hominis*. All these are, apparently, regarded as valid and distinct species. It is a long list—too long, in the reviewer's opinion—and it is impossible to discuss, in this place, all that is said about these various organisms. The following two main points must, however, be noticed.

First, it will be found that the descriptions of some of these flagellates are conspicuously defective in certain important particulars. For example, in the accounts of *Giardia* and *Chilomastix* no mention is made of the size of these organisms, nor of the number and attachments of their flagella—points of capital importance in the description of any flagellate. The figures alone—including one of *Giardia* (Pl. 1, fig. 3) labelled "*Craigia enterica*"—can hardly be regarded as adequate substitutes for descriptions, and would surely puzzle anyone unfamiliar with these creatures.

Secondly, it appears to the reviewer that these deficiencies on the zoological side are by no means counterbalanced by the comparative wealth of medical detail which is offered. Indeed, on this side the descriptions appear to exceed, both in length and in precision, our present real knowledge of the subject. The authors appear to have compiled these sections in a spirit of almost incredible credulousness. It is clearly unwise to treat the observations and interpretations of all workers as though they were all equally accurate and trustworthy, and to attempt, without analysis and criticism, to add error and truth together. The inevitable

result of such a benevolent and uncritical attitude towards everybody's evidence is particularly striking in the section on "*Craigia*" and "Craigiasis." A critical examination of the available evidence has convinced the reviewer—and many others—that "*Craigia*" is a mythical creature. Nevertheless, this supposed organism is here resuscitated—apparently without any misgivings—and over three pages are devoted to it and to the disease which it is alleged to cause. It is even regarded as "one of the most important flagellate infections of the human intestine." But, notwithstanding the detailed descriptions here given of the distribution, pathology, pathogenesis, symptoms, complications, and treatment of "Craigiasis," it can hardly be doubted that the cautious protozoologist will still feel entitled to inquire whether, in the first place, "*Craigia*" has any existence at all: and upon the answer to this question he will base his valuation of the literature dealing with "Craigiasis." It is remarkable moreover, that in their account of "*Craigia*" the present authors include a flagellate which is apparently new. It possesses a single flagellum, and forms a small uninucleate cyst. Why it should be inserted in the life-cycle of "*Craigia*"—described by its discoverers as a rhizopod—is by no means evident; nor can it be regarded, on the evidence presented, as lending any kind of support to the very questionable accounts of the "genus" previously published.

From the list of species already given it will be seen that *Tricercomonas intestinalis*, *Diplocercomonas soudanensis*, and *Enteromonas hominis* are all duly enrolled here as distinct organisms, belonging to three separate genera and species. In the reviewer's opinion, the three names are, in all probability, merely synonyms—three names for one and the same species. "*Tetrachilomastix intestinalis* (Sangiorgi) 1917" appears to be the flagellate named "*Tetratricomastix*" by SANGIORGI:* but why it should be thus transferred to FONSECA's subgenus *Tetrachilomastix* is not explained. In the opinion of the reviewer the organism was probably not an intestinal flagellate at all, but a coprozoic species of *Tetramitus*—probably identical with ARAGÃO's "*Copromastix*."†

The new generic name "*Ditrichomastix*" is here proposed for the organism called "*Octomitus hominis*" by CHALMERS and PEKKOLA.‡ It will be generally agreed that the introduction of new names in works of this character is undesirable: but apart from this, it must be noted that there is good reason to doubt whether the organism itself really represents an independent genus—or even a distinct species. It is a very questionable form.

Scattered through the article there are many minor errors, or inexact statements. We may note, as examples, that the free (*i.e.*, unencysted) form of *Giardia* is termed (p. 370) its "*free living* stage"—a misleading expression which sounds strangely in the mouth of a zoologist. Again, the sucker of the same flagellate is incorrectly called a "cytostome" (or rather, by an evident misprint, a "cystome," while its margin is termed the "cystostomal rim"). The statement on p. 371, repeated on p. 372, that this flagellate was recorded by British workers more frequently in dysenteric than in non-dysenteric cases, is hardly a correct representation of the facts: and the assertion (p. 382) that the flagellate originally discovered by LEEUWENHOEK in his own stools in 1687 (a mistake for 1681) was probably *Trichomonas*, is one which the reviewer has elsewhere been at some pains to disprove.§ A remark, on p. 388, that "in cases of irruption of hepatic abscesses of amebic origin into the respiratory region almost any intestinal flagellate might accompany the infection," seems to imply some novel conceptions regarding the pathology of amoebiasis: but it is doubtless in agreement with the authors' belief—which few readers of the *Bulletin*

* See this *Bulletin*, Vol. 10, p. 251.
† *Ibid.*, Vol. 8, p. 16.
‡ *Ibid.*, Vol. 8, p. 134.
§ *Ibid.*, Vol. 16, p. 89.

are likely to share—that *Trichomonas* is frequently found in liver abscesses (p. 367).

The article concludes with an enumeration of thirteen "Flagellates of minor importance." These turn out to be merely a number of coprozoic organisms, catalogued with little attempt at critical determination. Among them we find such forms as "*Bodo asiaticus*," "*B. cruzi*," "*B. italicus*," "*B. javanensis*," "*B. urinarius*," "*B. weinbergi*," and "*B. stercoralis*"—an impressive list, but one which has now lost some of its terrors for those who, like the reviewer, regard all these names as mere synonyms of the long-familiar free-living species *Bodo caudatus* (Dujardin) Stein. But whether this be right or wrong, it is surely no longer possible for any up-to-date protozoologist to accept all these debatable organisms as valid species. It seems to the reviewer regrettable, moreover, that no real attempt is anywhere made to discriminate between the free-living and the entozoic species, nor to draw any clear lines of demarcation between parasitic, commensal, and coprozoic forms. This, in itself, has already introduced much confusion into a subject wherein none exists in nature.

Only a few references are given at the end of the article, though many authors are referred to by name and date in the text—not always correctly (*e.g.*, "Lanfranchini," p. 366, a euphonious blend, apparently, of the two Italian investigators LANFRANCHI and FRANCHINI). But it is not very helpful, when one wishes to verify an author's statements, to be put off with such references as "Stiles (1915 a)" or "(1915 b)", which doubtless have a meaning for the authors themselves, but convey little to the world at large.

In conclusion, the reviewer will only say, for himself, that he has studied this work with increasing disappointment—amounting, in the end, almost to despair. The hopes with which he set out have not been fulfilled. He finds it difficult indeed to persuade himself that the authors can have devoted to the composition of the article that critical care and attention to detail which we have a right to expect of them, and which the subject itself merits: and he refuses to believe that the article, as a whole, accurately reflects the views of the most expert protozoologists in the United States at the present day.

Clifford Dobell.

DOBELL (Clifford) [M.A., F.R.S.] & O'CONNOR (F. W.) [M.R.C.S., L.R.C.P., D.T.M. & H.]. **The Intestinal Protozoa of Man.**—ix + 211 pp. With 8 plates and 2 figs. in text. 1921. London: Published for the Medical Research Council by John Bale, Sons & Danielsson. [Price 15*s.* net. Postage 1*s.*]

This admirable treatise, where a wealth of exact fact in accurate historical perspective is mirrored in a literary and artistic medium of much refinement, is meant to serve both the neophyte in protozoology whose circumstances merely necessitate a sound knowledge of intestinal protozoa, and the man of surer attainments whose purpose is higher mounted.

In a particularly well-delivered prologue we are introduced—and no one has qualified himself at greater pains than Dobell for the part of herald—to the original worthies of the piece: the illustrious Antony VAN LEEUWENHOEK, Father of Protozoology, who in 1681 discovered and described the little flagellate now known, through Mr. Dobell's own laborious researches, as *Giardia intestinalis*; DAVAINE (1854), who at a long interval brought two more flagellates out of the human *cella*; MALMSTEN (1856), the discoverer of *Balantidium coli*; KJELLBERG (1860), who first encountered the organisms now known as Coccidia in the human bowel; and Timothy LEWIS (1870) and D. D. CUNNINGHAM (1871), who first detected amoebae in human stools. By these elegant twofold introductions we are led into the classification of the Protozoa as a union of four great stocks or phyla—Rhizopoda, Mastigophora, Sporozoa and Ciliophora—which are all concisely defined.

In this prologue, too, the comparatively simple life-history of the intestinal protozoa is surveyed and brought into the practical perspective. The active period of freedom—or, at least, of active vitality—within the bowel is contrasted with the period of passive vitality or rest in a cyst outside the body, the one period quickly ending in death if the protection of the host's body is lost, the other being capable of persistence under suitable conditions of moisture for several weeks. Since the natural fate of the cyst is to pass out of the host's body, Dobell objects to the expressions "infected with cysts" and "cyst-carriers"; but this is really a case of thinking too precisely on the event, since these terms, if they are not sterling, are current counters acceptable to common sense: "medication which will kill the cysts in preference to the active forms," however, is villainous, and shows a most pitiful ignorance in him that uses it, and everyone will share the authors' disapproval of that phrase.

In considering the relations of the intestinal protozoa to their host the authors make it very clear how and why *Entamoeba histolytica*, *Balantidium coli*, and the Coccidia are true parasites, and how and why *E. coli* is not; but in their most laudable desires to distinguish between parasitism and other kinds of mutual association which are harmless and non-parasitic, and to emphasize the view that most of the intestinal protozoa of man should be placed in the latter category, they rather confound the plain man's powers of perception by classing *Giardia intestinalis* apart as a "food-robber"—by much the same argument as Jack Easy's wet-nurse used to extenuate her equivocal maternity, that it was "only a little one" and therefore not very damaging to reputation. But when an organism—if only a very little one—sits in a man's duodenum and appropriates the man's chyme—what can it be called but a parasite sans phrase?

The very fine study of the intestinal amoebae is prefaced by some general remarks, where, among other things, attention is directed to those points—nuclear structure and cysts—which most plainly exhibit the specifically distinctive features. All the five species are clearly and critically described in their active and passive modes of existence, the synonymy of each is given, and the account is rounded off with a tabular synoptical summary. With all this lucid description, and the beautifully executed accompaniment of figures, it is difficult to imagine even a beginner going astray.

In the chapter on Amoebiasis, which, of course, relates exclusively to the doings of *E. histolytica*, the help and criticism of Captain S. R. Douglas are acknowledged. Here *E. histolytica* is defined as a tissue-parasite invading the wall of the gut by a process of cytolysis. The authors conceive the amoeba as always—even in the very numerous cases, occurring in all parts of the world, where its presence in the gut causes so little inconvenience to its victim that the fact of its existence is entirely unsuspected—*always* at work destroying cells and eroding the mucous membrane, to ulceration. But "as a rule, the damage done to the gut wall of the host is compensated by regeneration on the part of the tissues. These are able to keep pace with the inroads of the amoebae, and a condition of equilibrium is thus established between host and parasite. Such a state of equilibrium must be regarded as the 'typical' or 'normal' condition in *E. histolytica* infections." We are, in short, to imagine the infected person as being in the position of Prometheus, with amoebae perpetually gnawing at his gut and the gut renewing itself as fast as it is eaten away—but unlike Prometheus in suffering no torments during the process.

How and why this golden age of "harmony"—if that be harmony where the guest must always "produce a more or less pathological condition of the colon of its host"—degenerates into an iron age of discord and suffering is not explained: it is merely stated that in a certain proportion of cases "pathological conditions," instead of the typical or normal more or less pathological condition of destruction and regeneration, result.

The authors' explanation of the now well-established fact that infection with *E. histolytica* is consistent with perfect health may be the right one, but, after all, it rests upon assumption. It is an equally rational assumption

that in a healthy gut under normal conditions the amoebae might not be able to make way against living tissues, and might maintain an existence—hard and penurious for themselves and harmless to their host—on the exuviae of tissue : and if it be true that the therapeutic action of emetine is due to its effect on the intestinal mucosa, rather than on the amoebae, this assumption—namely, that the often harmlessness of the amoebae is due to the resistance of the tissues—becomes plausible enough to encourage those perhaps old-fashioned pathologists to whom the conception of an invading organism, of common occurrence, which is always eroding and ulcerating the lining of the colon, but generally does no harm, seems very strange—and all the more strange because in another place the authors do admit the possibility of some kind of natural resistance to *infection*, and a resistance which differs in individual degree.

Apart from these rather questionable pathological prolegomena, this chapter on amoebiasis as a full-blown entity contains a wonderfully clear account both of the pathological phenomena themselves and of their correlations with the parasite and its life-cycle. The practical bearings of the facts are also very appositely expounded : that a person in the acute stage of bloody evacuations with active amoebae in them is not infective, since the evacuated amoebae must quickly perish ; that infection is brought about by swallowing cysts ; that cysts, which are the essence of infection, are passed not only by convalescents from amoebic dysentery, but also from many persons classed as healthy because, though carrying a dormant amoebic infection, they know nothing whatever about it ; that amoebic infection is extremely persistent, so that a convalescent carrier is long liable to relapses of dysentery and to other issues of amoebiasis, and a healthy unconscious carrier is liable to fall a victim to acute dysentery and other forms of amoebiasis at any moment ; all these lessons are admirably enforced, along with the text that probably between seven and ten per cent. of the population of Britain and a much higher percentage in the tropics is infected with *E. histolytica*.

The higher percentage of infection in the tropics is probably one of many illustrations of the imperfect control yet exercised by man over what Hobbes styles " meer Nature " in tropical countries (outside the large cities modelled on those of Western Europe), and of the small impression yet made on native tropical communities by our sanitary ideals. The enormously higher percentage of the pathological manifestations of infection in the tropics is a matter which the authors think not worth discussion at present. Here, however, few medical men will agree with them. It is *the* important matter ; and now that the specific limits of discussion are in this very volume so clearly defined and summarized and illustrated, for all men to comprehend, one of the greatest obstructions to useful discussion is removed.

In succeeding chapters the intestinal flagellates, coccidia, and ciliates are severally described, critically examined both in themselves and in their often complicated synonymy, and discussed in their ascertained and conjectural pathological bearings, in the same methodical way as the intestinal amoebae.

In the section on the flagellates—where *Embadomonas intestinalis* and *Enteromonas hominis* are not treated with quite the same sure and firm touch as the other species—the authors adhere to the opinion that " there is as yet no good evidence to prove that any intestinal flagellate found in man is pathogenic, but that there is very considerable evidence to show that most and probably all of them are harmless."

The section on coccidia and coccidiosis is appropriately prefaced with an account of the typical coccidian life-cycle, since specific distinctions are drawn from the oocysts and their contents, and the (probably transient) nature of a coccidian infection in man can only be inferred from general knowledge of the life-cycle acquired independently of man. Attention is directed to the fact that the coccidia of man, in their present geographical distribution seem to be more domiciled in tropical and subtropical latitudes, and that the species most frequently found in man, *Isospora hominis*,

seems to have a centre in the countries of the Eastern Mediterranean region.

In the section on ciliates attention is concentrated on *Balantidium coli* and its pathological effects. *Balantidium minutum*, of which the authors admit only two authentic cases of infection, and *Nyctotherus faba*, of which they allow only one, are also figured and briefly described. Here, too, attention is very fitly given to a considerable number of ciliates that have been found at various times in human faeces and have even been invested with pathogenic attributes, but which have probably been accidentally introduced in the water or saline solutions used in manipulating stools. Some of these are rash identifications and are entirely questionable; others, such as Chilodon and Colpoda, are free-living forms of common occurrence. They are not to be confused either with the intestinal protozoa or with the Coprozoic fauna presently to be noticed.

It is difficult to overpraise the section on the diagnosis of intestinal protozoal infections, or to estimate its probable effect on the study of this branch of medical protozoology. I believe that only a man who knows what it is to live in an up-country station abroad, apart from professional associations, and has felt the bitterness of missing golden opportunities through technical inexperience or indexterity, can appreciate to the full this truly admirable section. Many such a man will, I feel certain, bless the name of Dobell. Here are to be found full and explicit directions for doing all that ought to be done and avoiding all that should be left undone in collecting material and preparing it for examination and treating it for permanent study; all the finger-posts and danger-signals; all the escort and attentive support of a chief scout.

In indicating common sources of error attention is drawn, among many other things, to living organisms which may be found in fairly fresh human faeces, but have not lived in the bowel nor been introduced in water or saline solution added to the stool afterwards. These organisms, which belong to the Rhizopoda and Mastigophora, are treated in a separate section, as the "Coprozoic Protozoa of Human Faeces."

The "Coprozoic" forms are free-living protozoa found naturally in water or decomposing organic matter. They may make their way into human faeces just as they do into organic infusions exposed to the air; or, in the encysted form, they may be swallowed in food or drink, and if the cysts survive the passage through the body they may excyst and proliferate in the evacuations: that they do not excyst within the gut is explained as probably due to the too-high temperature or to insufficiency of oxygen. The species of most common occurrence in human faeces are here described, and ten of them are figured: they include three species of amoebae, one shell-secreting amoeba, two species of Bodo, two of Cercomonas, one Copromonas and three other flagellates. This also is a most valuable section, and should prevent the discovery of many mares' nests.

An infinity of care and skill is embodied in the plates. One of the most happily conceived is Plate VIII, a triptych, the panels of which are identical in everything but the accessory detail of the constituent figures, displaying the cysts of the chief intestinal protozoa of man. In one panel they appear as when alive, in the next as when seen in iodine solution, and in the third as when fixed and stained with iron-haematoxylin. The figures are magnified 2,000 diameters; they are not ideal in the sense of being either visionary or diagrammatic, and they are not real (if one may so strain the term) in the sense of being either photographic or actual portrayal: their author calls them "semi-diagrammatic"—a definement by which they suffer perdition: they, rather, represent the distilled essence of a multitude of visual judgments, and are perhaps best described in the vernacular as "just It."

Since in the preface the principal author complains of sometimes being taken to task for not having sought to find out acceptable words for the

errors of other workers, it may be stated, in conclusion, that in the present volume no withers are wrung: the author may with a clear conscience say, like Descartes, "My design was solely to find assured certainty, and to throw aside the loose earth and sand so that I might reach the rock or the clay beneath."

A. Alcock.

CAULLERY (Maurice). **Le Parasitisme et la Symbiose.** (Encyclopédie Scientifique: Bibliothèque de Biologie Générale.)—400 pp. With 53 text figs. 1922. Paris: Librairie Octave Doin: G. Doin, Editeur. [Price 14 fr.]

Commensalism, symbiosis and parasitism are mysteries of adaptive evolution which have always attracted the biologist, and have enthralled and chastened the philosopher even more completely than has the "flower in the crannied wall."

Everyone is familiar with instances of commensalism, where two organisms of different kind are bound together, without derogation of their individual beauty and power, in a common life of mutual benefit; of symbiosis, where two organisms of different kind are so mingled and merged in an unified existence that they become a differentiated "tertium quid"; and of parasitism, where, of two organisms of different kind always living in association, one uses the other entirely for its own selfish ends and usually suffers some loss of beauty in the consequence. The three ideas are perfectly distinct; and yet, by extensive observation and consideration of Nature, we undoubtedly can pass without a break from the ethically-beautiful idea of necessary reciprocity illustrated by the most perfect instances of commensalism (*e.g.*, Paguropsis and its adjustable overcoat of polyps), and the mechanically-beautiful idea of reciprocal necessity illustrated by the purest examples of symbiosis (*e.g.*, the algae and the fungi that compose the lichen), to the ugly perfection of a parasite such as Sacculina, which, in reducing its partner (ironically styled "host") to sexual impotence, itself becomes little better than a wart. And thus with Job we come, by a road of wonders and indirections, and almost incredible surprises, to the old conclusion that in Nature there are neither bounds and limits nor rights and wrongs, nor any beauties and uglinesses; but that these things exist only in the mind and heart of that most wonderful piece of work—Man.

In this book an immense number of instances of commensalism, parasitism, and other allied phenomena are collated and thoughtfully considered. The result may be commended to the medical profession, which, though not so free as the street-corner politician in its use of the term "parasite," is often inclined to ignore what may be called the alleviating and semi-alluring aspects of parasitism in the abstract. Particularly to be commended are Chapter X, where the reciprocal reactions of parasite and host are discussed; Chapter XI, dealing among other things with physiological aspects of symbiosis, and with hereditary symbiosis; Chapter XII, which leads up to the borderland between symbiosis and pathology; and Chapter XIII, which opens up speculations on the conception of cell-life and metabolism as a sort of microcosm of symbioses. In Chapter IX also there are some interesting discussions on the theory—with which the name of ROUBAUD is identified—of "biological prophylaxis" against insect "carriers" of disease.

A. Alcock.

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